

Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology

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Abstract

In the last decade, cardiac vagal tone has emerged as a psychophysiological marker of many aspects of behavioral functioning in both children and adults. Research efforts during this time have produced an extensive list of vagal tone correlates that includes temperamental variables as well as both anxious/internalizing and disruptive/externalizing behaviors. This potentially confusing state of affairs is compounded by developmental shifts in vagal tone–behavior relations that to date have not been elucidated. In this paper, the vagal tone literature is reviewed, and discrepancies, including the lack of specificity of vagal tone as a psychophysiological marker, are clarified. Such clarification requires that we (a) view vagal tone–behavior relations in developmental context, (b) juxtapose vagal tone–behavior relations in typical and atypical samples, and (c) consider the parasympathetic underpinnings of vagal tone as but one component in a broader model of autonomic nervous system functioning. Such a model is provided by combining Gray's motivational theory with Porges's polyvagal theory. Together these models account for behavioral and emotional differences in a diverse range of psychological disorders that are not differentiated by either model alone. Moreover, use of the integrated model offers a theory-driven approach to the study of autonomic nervous system–behavior relations.

Cardiac vagal tone has received considerable attention in recent years as a psychophysiological marker of emotion regulation, and of certain aspects psychological adjustment. In infant and child samples, individual differences in indices of vagal tone and vagal reactivity have been demonstrated to reflect emotional expressiveness (Stifter, Fox, & Porges, 1989), temperamental reactivity (e.g., Calkins, 1997), attachment status (Izard, Porges,

Simmons, Haynes, & Cohen, 1991), empathic responding (e.g., Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994), social competence (Eisenberg, Fabes, Murphy, Maszk, Smith, & Karbon, 1995), attentional capacity (Suess, Porges, & Plude, 1994), behavioral inhibition (e.g., Kagan & Snidman, 1991), and aggression (e.g., Pine, Wasserman, Coplan, Fried, Sloan, Myers, Greenhill, Shaffer, & Parsons, 1996). Additionally, studies of adolescents and adults have linked atypical vagal tone to hostility (Sloan, Shapiro, Bigger, Bagiella, Steinman, & Gorman, 1994), aggression (Mezzacappa, Tremblay, Kindlon, Saul, Arseneault, Seguin, Pihl, & Earls, 1997), depression (e.g., Carney, Saunders, Freedland, Stein, Rich & Jaffe, 1995), anxiety (Thayer, Friedman, & Borkovec, 1996), panic (Friedman, Thayer, Borkovec, Tyrrell, Johnson, & Col-

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umbo, 1993), bulimia (Kennedy & Heslegrave, 1989), anorexia (Petretta, Bonaduce, Scalfi, de Filippo, Marciano, Migaux, Themistoclakis, Ianniciello, & Contaldo, 1997), hypnotic susceptibility (Harris, Porges, Clemenson Carpenter, & Vincenz, 1993), and functional dyspepsia (Haug, Svebak, Hausken, Wilhelmsen, Berstad, & Ursin, 1994). Thus, the correlates of vagal tone and vagal reactivity include a broad range of psychological adjustment variables, both adaptive and maladaptive, that span developmental stages from infancy to adulthood. This pattern supports the assertion that the biobehavioral system indexed by vagal tone subserves core regulatory functions implicated in the development and maintenance of both normal and pathological behavior patterns (Porges, 1996; Porges, Doussard-Roosevelt, & Maiti, 1994).

However, the breadth of vagal tone correlates also raises questions about the specificity and thus utility of the index as a marker of psychological functioning, as do several inconsistencies represented in the literature, many of which are well replicated. As the above list suggests, reduced vagal tone reflects both externalizing disorders, such as antisocial behavior and aggression, and internalizing disorders, such as anxiety and depression. Moreover, vagal tone is a positive correlate of behavioral reactivity and temperamental difficulty in infancy, yet a negative correlate of similar measures during the preschool years (Porges, Doussard-Roosevelt, Portales, & Suess, 1994). Vagal tone has also been interpreted as an index of both emotion regulation (see Porges et al., 1994), which is often presumed to be socialized, and temperamental reactivity (Calkins, 1997; Stifter & Fox, 1990), which is largely inherited (Campos, Barrett, Lamb, Goldsmith, & Stenberg, 1983).

These apparent disparities suggest that relations between vagal tone and psychological adjustment are more complex than has often been supposed. Indeed, in the history of scientific inquiry similar paradoxes have often precipitated theoretical shifts that confer a greater level of understanding of complex systems. Planetary motion, for example, could not be explained sufficiently before the recognition

that the Earth is a noncentral component of a larger solar system. Although the implications here may be less profound, it will be suggested in the sections to follow that many of the disparities in the vagal tone literature are clarified when the vagal system is viewed as a noncentral component of the autonomic nervous system. It will further be suggested that these findings reflect a coherent and meaningful pattern when viewed from the perspective of developmental psychopathology.

Tenets of Developmental Psychopathology

The developmental psychopathology perspective was formalized by Sroufe and Rutter (1984), who outlined several broad tenets that set the field apart from related scientific disciplines. First, developmental psychopathologists are concerned with outlining the course of disorders at a greater level of specificity than epidemiologic description. Thus, they wish to relate the emergence of disorders to specific vulnerabilities, both biological and psychological, that compromise one's ability to negotiate developmental milestones successfully. Because of the assumption that behavioral and emotional repertoires differentiate hierarchically over time, with success at one developmental period laying the groundwork for future successes (Werner, 1957), identifying such vulnerabilities is considered an essential step toward a comprehensive understanding of the etiology and course of most disorders (see Cicchetti, 1990).

Second, maladaptive development can be fully understood only when juxtaposed with adaptive development. To the extent that pathological development represents deviation from optimal functioning, both typical and atypical developmental trajectories must be mapped and compared in order to properly conceptualize, diagnose, and treat disorders (Cicchetti & Toth, 1998). A corollary of this tenet is that studying biological and psychological variability in normative samples may not be informative with respect to high-risk groups, both because the number of deviant cases is typically insufficient to generalize from and because differences in develop-

mental trajectories may be washed out by the large proportion of normative cases.

Third, Sroufe and Rutter (1984) suggested that the processes and structures involved in both normal and atypical development are likely to be complex. This sentiment has been echoed and elaborated on by others (e.g., Cicchetti & Toth 1998; Kagan, 1997; Thelen & Smith, 1998), who have called for more thorough assessment of multiple biological and psychological determinants of behavioral development. According to this tenet, no single biological system, such as that indexed by vagal tone, can be expected to contribute toward a meaningful understanding of behavior, either adaptive or maladaptive, since behavior is the product of multiple systemic inputs.

In this paper, the potential contribution of these principles toward clarifying the diverse pattern of empirical correlates of vagal tone and vagal reactivity will be presented. As the following literature review will reveal, most of the research on psychological adjustment and vagal tone has been descriptive, and thus guided little by developmental theory. Although the polyvagal theory of Porges (1995) has been influential, it is largely a phylogenic and functional account of the vagal system and includes limited elaboration on developmental or systemic considerations. Where development is considered, it is primarily in relation to the integrity of the vagal system in determining health outcomes vis-à-vis high-risk infants (see Porges, 1996). Moreover, while the theory represents a significant step forward in our understanding of the contribution of the autonomic nervous system to both adaptive and maladaptive behavior, it speaks primarily to parasympathetic functioning. Thus, the theory implicitly minimizes the role of the sympathetic nervous system, and of possible sympathetic-parasympathetic interactions, in affecting behavior.

In the sections to follow, polyvagal theory will be briefly outlined, and the literature regarding vagal tone and vagal reactivity reviewed, from infancy through adulthood. Empirical relations which are not well accounted for without considering developmental processes will be highlighted. Finally, the utility of combining Porges's theory of parasympa-

thetic functioning with Gray's (1982a, 1982b, 1987a, 1987b) motivational theory will be presented. Before doing so, however, it is necessary to define and operationalize vagal tone and vagal reactivity, which are assessed by measuring heart rate variability.

Heart Rate Variability and Parasympathetic Cardiac Control

The autonomic nervous system (ANS) comprises the sympathetic (SNS) and parasympathetic (PNS) branches, both of which innervate most internal organs and bodily systems. The effects on a target organ of SNS and PNS activation are generally antagonistic. Patterns of cardiac activity, for instance, are determined primarily by the dynamic interaction of acceleratory SNS activation and deceleratory PNS activation (see Berntson, Cacioppo, & Quigley, 1993; Berntson, Cacioppo, Quigley, & Fabro, 1994). The deceleratory parasympathetic component is provided by the vagus, or tenth cranial nerve.

The vagus nerve includes both efferent and afferent fibers, so neural traffic through it is bidirectional. Efferent fibers originating in the brain stem terminate on the sinoatrial (SA) node, which serves as the cardiac pacemaker. Because vagal efference is inhibitory, activity through these fibers slows heart rate by decreasing SA node firing (see Levy & Warner, 1994). Vagal afferent fibers, on the other hand, originate in the heart and project to the nucleus tractus solitarius (see Porges, Dousard-Roosevelt, Portales, & Greenspan, 1996). These fibers provide continuous feedback to the brain, facilitating regulation of cardiac functioning (see Porges et al., 1996).

Vagal tone is typically estimated from respiratory sinus arrhythmia (RSA), or the degree of ebbing and flowing of heart rate during the respiratory cycle (Berntson, Bigger, Eckberg, Grossman, Kaufmann, Malik, Nagaraja, Porges, Saul, Stone, & van der Molen, 1997; Hayano, Sakakibara, Yamada, Yamada, Mukai, Fujinami, Yokoyama, Watanabe, & Takata, 1991; Katona & Jih, 1975). RSA results from increases in vagal efference during exhalation, which decelerate heart rate, and decreases in vagal efference during inhalation,

which accelerate heart rate. Heart rate cannot be used to assess vagal tone directly, however, because acceleratory sympathetic projections to the SA node also contribute to chronotropic (rate-related) cardiac activity (see Randall, 1994). Thus, measures of RSA have been sought that are devoid of sympathetic influences. Although consensus regarding the most preferable method has only begun to emerge (see Berntson et al., 1997), all such measures index heart rate variability (HRV), or beat to beat differences in the length of the cardiac cycle over time.

The simplest methods of assessing HRV involve calculating descriptive statistics on series of electrocardiographic (ECG) R waves. Such indices, including standard deviation, variance, and mean successive difference, correlate highly with one another, and with vagal efference as assessed via pharmacologic blockade (Hayano et al., 1991). Concern has emerged over the use of these measures, however, both because of failures to replicate fully the results of blockade studies (Grossman, Karemaker, & Wieling, 1991)¹ and because RSA accounts for only about half of observed HRV (Grossman, van Beek, & Wientjes, 1990). Thus, alternative indices have been developed that statistically eliminate influences on HRV that are extraneous to RSA. Examples include Porges's moving polynomial algorithm (Porges, 1986), which has been used extensively in infant and child work, and Grossman's peak-valley technique (Grossman et al., 1990), which has more often been applied with adults. Although it is be-

yond the scope of this paper to review these methods in detail, evidence suggests that estimates of RSA derived from each are highly correlated with one another, and with the descriptive approaches previously outlined (Grossman et al., 1990). In addition, the Porges method is highly correlated with descriptive measures of heart period variance in infants (Izard et al., 1991). It should be noted, however, that convergence of these alternative indices may be dependent on statistical control of respiration rate (Grossman et al., 1990), which is inversely related to RSA (see Berntson et al., 1997; Grossman et al., 1991).

In addition, Grossman and colleagues (1991) have demonstrated that changes in respiration rate and tidal volume influence RSA independent of vagal tone. This point deserves elaboration because RSA and vagal tone have been treated as equivalent in much of the literature to be reviewed. Although correlations between vagal tone (as assessed via pharmacologic blockade) and RSA can approach .9 during paced breathing (Hayano et al., 1991), and when respiration rate and tidal volume are statistically controlled (Grossman et al., 1991), these correlations drop to between .5 and .7 in the absence of such controls (Grossman et al., 1991; Grossman & Kollai, 1993). Thus, RSA is an imperfect index of cardiac vagal tone (see also Jennings & McKnight, 1994), so the terms should not be used interchangeably. Moreover, few attempts to control for respiration have been included in studies involving children, providing a potential confound for many extant findings, an issue that will be revisited in later sections of this article.

Additionally, although the more advanced techniques described thus far can eliminate or reduce influences on HRV that are not attributable to RSA, none can resolve those influences. This has prompted many researchers to turn to spectral analysis in their assessments of vagal tone. Spectral analysis involves the decomposition of heart rate time series into component frequencies through Fourier transformations. These components are divided into low-frequency variability (less than 0.04 Hz), midfrequency variability (0.04–0.15 Hz), and high-frequency variability (greater than

1. Two methods of vagal tone assessment via pharmacologic blockade are represented in the literature. Both involve the administration of a beta-adrenergic antagonist, which eliminates sympathetic nervous system influences on heart rate. In the simpler method (e.g., Grossman et al., 1991), vagal tone is taken as the difference between preblockade heart period, which is sympathetically and parasympathetically mediated, and postblockade heart period, after sympathetic effects have been removed. In the second method (e.g., Grossman & Kollai, 1993; Hayano et al., 1991) a parasympathetic antagonist is also administered, and the reduction in HRV between the beta-adrenergic and the parasympathetic blockers is used to represent cardiac vagal tone. This method also eliminates nonneural influences on HRV from vagal estimates.

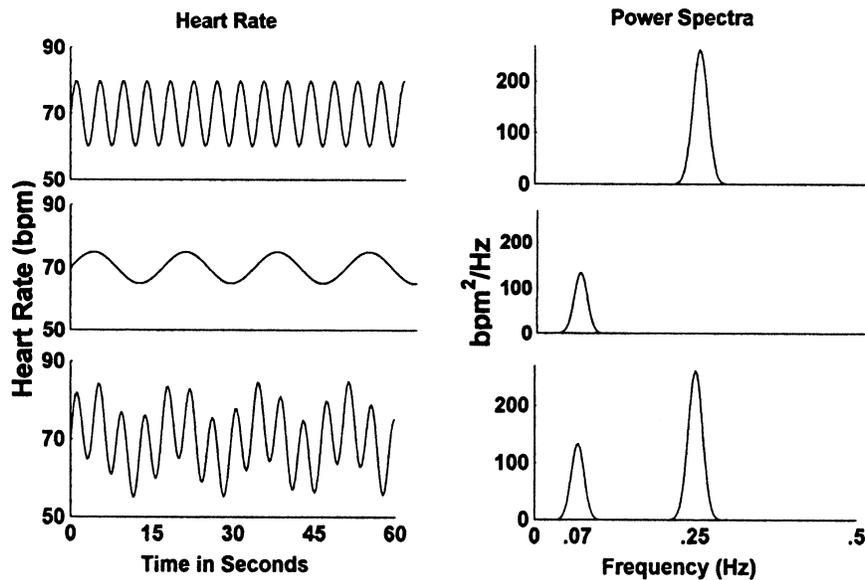


Figure 1. Fictitious heart rate signals and associated power spectra. The top panels represent pure high-frequency variability (0.25 Hz), as associated with RSA. The middle panels represent low-frequency variability (0.07 Hz), primarily of sympathetic and nonneural origin. The bottom panels represent the combined signal including both high- and low-frequency components. Actual heart rate signals include spectral power at additional frequencies (adapted from Mezzacappa et al., 1994).

0.15 Hz; see Mezzacappa, Kindlon, Earls, & Saul, 1994; Mezzacappa et al., 1997). Pharmacologic blockade studies have suggested that sympathetic influences on HRV are confined to the low and midfrequencies, whereas parasympathetic influences, including RSA, are observed primarily in the high-frequency range (Akselrod, Gordon, Ubel, Shannon, Barger, & Cohen, 1981; Akselrod, Gordon, Madwed, Snidman, Shannon, & Cohen, 1985; Berger, Saul, & Cohen, 1989; Pomeranz, Macaulay, Caudill, Kutz, Adam, Gordon, Kilborn, Barger, Shannon, Cohen, & Benson, 1985; Saul, Berger, Chen, & Cohen, 1989; Saul, Berger, Albrecht, Stein, Chen, & Cohen, 1991). Sample heart rate time series and associated spectra are presented in Figure 1. The area under a given peak represents the power, or amplitude, of heart rate oscillation at that frequency band. In the example pictured, spectral power in the respiratory (high-frequency) range, which represents vagal influences, is roughly double that in the low-frequency range, where sympathetic and nonneural influences predominate. This illustrates

how contributions to HRV outside the RSA band can be resolved using spectral analysis.

Regardless of the method employed, the assumption underlying the assessment of RSA is that, as a peripheral measure of ANS activity, it serves as a proxy for more central regulatory processes that cannot be measured non-invasively (Porges, 1996). The origins of these regulatory processes, both at neuroanatomical and phylogenetic levels of analysis, are outlined in Porges's (1995) polyvagal theory.

Polyvagal Theory

Porges (1995) specified two sources of vagal efference to the heart, one originating in the dorsal motor nucleus and the other in the nucleus ambiguus, and both terminating on the SA node. The dorsal motor nucleus controls what Porges refers to as the vegetative vagus, which mediates reflexive cardiac activity, including the deceleration of heart rate associated with orienting. This vagal branch is phylogenetically older, and presumably rooted in

the primary coping strategy of reptiles, which freeze when threatened.

In contrast, the smart vagus, which originates in the nucleus ambiguus, is distinctly mammalian, and mediates cardiac activity when environmental demands require extra-reptilian coping. After orienting, mammals must either attend to and engage with the initial threat or resort to fight-flight responding. Engagement requires sustained attention, which is accompanied by vagally mediated inhibition of heart rate (e.g., Suess et al., 1994; Weber, van der Molen, & Molendaar, 1994). Alternatively, fighting and fleeing are accompanied by rage and panic, respectively, which are characterized by near complete vagal withdrawal and large sympathetically mediated heart rate accelerations (George, Nutt, Walker, Porges, Adinoff, & Linnoila, 1989; see Porges, 1995, 1996). Thus, the association between intense emotional experience and vagal withdrawal is functional, facilitating bursts of metabolic output in situations of danger.

The assertion that vagal outflow from the nucleus ambiguus is functionally linked to attentional and emotional processing is further supported by structural characteristics of the mammalian brain stem. The nucleus ambiguus also innervates the larynx, which is used by most mammals to produce vocalization, the primary means of communicating emotional state. Additionally, the facial and jaw muscles, which are implicated in both vocal and nonvocal expressions of emotion, are innervated by the trigeminal and facial motor nuclei, adjacent to the nucleus ambiguus. The facial motor nucleus also innervates the inner ear, which is implicated in both attentional and communicative processes. Moreover, the actions of all of these source nuclei are coordinated in concert by the reticular formation (Butler & Hodos, 1996). Taken together, these structural characteristics lend support to the assertion that vagal regulation is linked to attentional and emotional processing.

The empirical literature generally supports this assertion, although relations between vagal outflow, attention, and emotion are complex. Tonic indices of RSA obtained during periods of relative quiescence, for example,

appear to reflect temperamental reactivity and emotionality. In contrast, shifts in RSA in response to environmental demands appear to reflect attentional focus, emotion regulation, and mood state. A potential source of confusion in the literature to be reviewed is the use of the term vagal tone to refer to (a) tonic measures of RSA, (b) reactivity measures of RSA, and (c) Porges's (1986) moving polynomial method of RSA quantification. These alternative usages present two problems. First, as previously mentioned, the validity of RSA as an index of vagal tone is moderate when respiration is not controlled. Thus, in the present article, the term vagal tone will be reserved for theoretical discussion and will not be used to refer to any indices of RSA. Second, referring to both tonic RSA and RSA reactivity as vagal tone masks important distinctions in the aspects of functioning marked by each. Thus, RSA and RSA reactivity will be treated separately in an effort to clarify these distinctions. It is to the empirical relation between tonic RSA and temperament that this article now turns.

RSA and Temperament

Infant studies

Rothbart and Bates (1998) define temperament as "constitutionally based individual differences in emotional, motor, and attentional reactivity and self regulation" (p. 109). According to this definition and others, a central component of temperament is emotionality (see Gunnar, 1990), which refers to individual differences in displays of positive and negative affect in response to environmental demands (Buss & Plomin, 1975). Historically, work with infants has focused more on negative emotionality, which has been referred to as anger proneness (Goldsmith, 1996), distress to limitations (Rothbart, 1981), fussiness-difficultness (Bates, Freeland, & Lounsbury, 1979), irritability (Sanson, Prior, Garino, Oberklaid, & Sewell, 1987), irritable distress (Rothbart & Mauro, 1990), and negative mood (Carey & McDevitt, 1978). Infants high on negative affectivity are apt to respond to environmental stressors with marked pro-

test, including behavioral reactivity and crying.

Much of the work by Porges and colleagues suggests that RSA is a psychophysiological marker of behavioral reactivity and emotionality in infancy. High RSA infants subjected to a pacifier withdrawal procedure, for instance, cried more than their low RSA counterparts (Stifter, Fox, & Porges, 1986). In response to circumcision, newborn males with high RSA exhibited greater pain reactivity, as assessed by heart rate acceleration and fundamental cry frequencies (Porter, Porges, & Marshal, 1988). Additionally, in a sample of premature infants DiPietro and Porges (1991) reported greater behavioral reactivity for high RSA neonates in response to a feeding procedure requiring a tube run through the nose or mouth. Moreover, newborns with higher RSA have exhibited larger cortisol responses to a heel-stick procedure for drawing blood, suggesting greater stress reactivity (Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995).

High RSA at 9 months also predicts maternal ratings of temperamental difficulty (Porges et al., 1994), as assessed by the Infant Characteristics Questionnaire (Bates et al., 1979). In two additional studies, 5-month-old infants with high RSA displayed more negative reactivity to a standardized procedure in which their arms were restrained by their mothers (Fox, 1989; Stifter & Fox, 1990). Ratings of emotional expression were derived from Ekman and Friesen's (1984) EM-FACS coding system. Similar results were obtained by Calkins and Fox (1992), who reported greater RSA in 5-month-old infants who cried in response to the presentation of novel geometric patterns, and to pacifier withdrawal, than in infants who cried in response to one or neither of the events.

These findings imply that high RSA in infancy is a marker of negative emotionality and difficulty. Yet RSA also predicts positive affective expression in infants and may therefore index general behavioral and emotional reactivity rather than negative emotionality alone. In the study reported above, Fox (1989) obtained a near significant relation ($p = .06$) between infant RSA and EM-FACS-coded positive reactivity during a peek-a-boo procedure

with mothers and strangers, despite limited power due to a small sample size. Moreover, the finding of greater positive facial expressivity was replicated by Stifter et al. (1989), using Izard and Dougherty's (1980) AFFEX coding system. Higher RSA infants exhibited more joy and interest expressions toward a stranger than did their lower RSA counterparts.

Finally, high RSA newborns are more responsive to a variety of environmental stimuli than are low RSA newborns. Porges, Arnold, and Forbes (1973), for example, demonstrated that infants with high as opposed to low baseline heart rate variability responded to the onset of a stimulus tone with greater heart rate acceleration, and to the offset of the tone with greater heart rate deceleration. Porges, Stamps, and Walter (1974) obtained similar findings when subjecting infants to changes in illumination. Moreover, only high RSA newborns exhibited conditioned heart rate deceleration in response to a tone stimulus that had been paired with a pattern of blinking lights (Stamps & Porges, 1975). Richards (1985a) reported that high RSA infants attended to novel stimuli for shorter periods of time than low RSA infants. Because a similar pattern was observed for older (20-week) as opposed to younger (14-week) participants, the result was interpreted as evidence for greater attentional capacity and processing speed in the high RSA group. Further support for this conjecture is provided by the finding that high RSA infants were less likely to be distracted, as assessed by gaze aversion toward a peripherally presented interrupting stimulus (Richards, 1987). Finally, Linnemeyer and Porges (1986) demonstrated that high RSA 6-month-olds looked at familiar stimuli for shorter periods, but at unfamiliar stimuli for longer periods, than did infants with lower RSA.

Taken together, these reports suggest that RSA reflects the capacity of infants to engage actively with the environment. Such engagement includes behavioral, attentional, and emotional responsiveness, both positive and negative, to external events and challenges. Indeed, Richards and Cameron (1989) found positive associations between RSA and the approach subscale of the Infant Temperament Questionnaire (Carey & McDevitt, 1978) at

ages 14, 20, and 26 weeks. Moreover, inhibited children who are low on approach and high on fear exhibit low RSA (Kagan, Reznick, & Snidman, 1987; Rubin, Hastings, Stewart, Henderson, & Chen, 1997), as do the socially and affectively inexpressive infants of depressed mothers (Field, Pickens, Fox, Nawrocki, & Gonzalez, 1995; Field, Lang, Martinez, Yando, Pickens, & Bendell, 1996).

Child studies

Associations between temperament and RSA also occur in 2- and 3-year-olds (Calkins, 1997), although alternative patterns begin to emerge. While individual differences in RSA (Porges et al., 1994; Porter, Bryan, & Hsu, 1995) and in indices of activity and reactivity (Worobey & Blajda, 1989) exhibit moderate to high stability after about 3 months of age, relations between RSA and behavior do not. This pattern is perhaps best demonstrated in the work of Porges and colleagues (1994), who as expected reported positive associations between RSA, behavioral reactivity, and maternal-rated temperamental difficulty in 9-month-olds, as assessed by Bates' (1984) Infant Characteristics Questionnaire. By age 3 years, however, the same 9-month RSA measure was a negative correlate of maternal-rated difficulty, after the relation between 9-month RSA and 9-month difficulty was statistically removed. Additional studies suggest that this finding is not anomalous. Premature newborns who exhibited relatively high RSA at birth, for instance, were rated as more socially competent at age 3 years on the California Preschool Social Competency Scale (Levine, Elzey, & Lewis, 1969) than were their low-RSA counterparts (Doussard-Roosevelt, Porges, Scanlon, Alemi, & Scanlon, 1997). Similarly, Fox and Field (1989) found that 3-year-olds with high RSA and a high activity level were better adjusted to preschool than their low-RSA peers, as assessed by the amount of interactive play they engaged in.

Compared to the infant literature, fewer studies have addressed the relations between RSA and attentional processes in preschool and grade-school children. Shibagaki and Fur-

uya (1997) reported reduced baseline RSA in 18 children with attention-deficit hyperactivity disorder (ADHD) compared to 49 controls. This study, however, was characterized by several methodological shortcomings, including imprecise ECG recordings, and failure to rule out comorbid conduct disorder, the importance of which will be presented in later sections of this paper. Suess et al. (1994) subjected fourth- and fifth-graders to a continuous performance task in which they were required to depress a keyboard space bar when a prespecified pattern of numbers was displayed. Children with higher baseline RSA performed better, as indexed by signal detection sensitivity, within the first of three 3-min blocks, than did children with low RSA. Hyde and Izard (1997), however, reported no relation between length of attention span and baseline RSA in a longitudinal study of 3-, 4-, and 5-year-olds. In that study, children watched a 5-min videotaped story while their gaze fixation, motor activity, frequency of speech, and facial expressions were monitored. None of the variables was related to RSA. These two reports represent the only studies addressing associations between RSA and attention in preschool children, thus constraining conclusions.

Eisenberg and colleagues have demonstrated in a series of experiments that high RSA predicts both expressions of empathy for others in distress and general social competence, particularly in boys. Kindergarten and second-grade children exposed to a crying infant, for instance, were more likely to talk to the infant and offer instrumental help if they exhibited high RSA (Fabes et al., 1994). Similarly, high-RSA third-grade boys exposed to a film of two frightened children were rated as more concerned, based on facial expressions of emotion (Fabes, Eisenberg, & Eisenbud, 1993). The high-RSA boys were also more likely to report subjective feelings of sympathy, less likely to become autonomically aroused (as assessed by skin conductance), and less likely to disengage by looking away. No significant relations were reported for girls. Moreover, in a sample of 6- to 8-year-olds, boys with high RSA were rated as more sociable by their teachers, and more

emotionally regulated by their parents, than were boys with low RSA (Eisenberg et al., 1995). Curiously, teacher-report findings were reversed for girls, and RSA was unrelated to any parent-report ratings. Similar sex differences in peer ratings of social competence have also been reported (Eisenberg, Fabes, Karbon, Murphy, Wosinski, Polazzi, Carlo, & Juhnke, 1996).

The sex effects in these three studies raise questions regarding the generalizability of RSA–behavior relations across gender. In part, the differences may be attributable to restricted ranges on all indices for girls. In the first analysis (Fabes et al., 1993), girls were rated as more sympathetic, in the second (Eisenberg et al., 1995) as more socially skilled and better regulated, and in the third (Eisenberg et al., 1996) as more prosocial. Moreover, although null findings have been reported (Fabes et al., 1993; Suess et al., 1994), several researchers have found higher RSA in girls than in boys (e.g., Fabes et al., 1994; Stamps & Porges, 1975) and in women than in men (see Lehofer, Moser, Hoehn–Saric, McLeod, Liebmann, Drnovsek, Egner, Hildenbrandt, & Zapotoczky, 1997). Thus, reduced variability in both RSA and in measures of social competence may have attenuated correlations. Indeed, girls' scores were characterized by lower variance than boys' scores on 29 of the 40 behavioral and emotional indices employed in these studies. Nevertheless, sex differences deserve further scrutiny, particularly given the above pattern, where disparities appear to become more marked with increasing age, and given that most researchers have ignored sex as a predictor of RSA.

Sex effects notwithstanding, changes in RSA from a marker of negative emotionality in infancy, to a marker of positive emotionality in toddlerhood, to a marker of social competence in preschool and grade school, cannot be accounted for without considering development. As outlined by others, the period from infancy to toddlerhood is one of dramatic emotional and behavioral differentiation (e.g., Cole, Michel, & O'Donnell Teti, 1994; Gunnar, 1990). Newborns have few means at their disposal for impacting upon their envi-

ronments beyond expressing displeasure, which is the predominant emotion in their affective repertoires for the first few weeks of life. Over the next year, as parasympathetic influence on cardiac control increases (Harper, Walter, Leake, Hoffman, Sieck, Serman, Hoppenbrouwers, & Hodgman, 1978; Katona, Franz, & Egbert, 1980; Richards, 1985b), expressions of positive emotion become more common, and expressions of negative emotion more differentiated, including displays of both anger and fear (Sroufe, 1979). During the 2nd year of life, positive affect differentiates, as emotions such as joy and excitement are observed. This developmental sequence, with negative emotions and reactivity emerging and differentiating before positive emotions, may be in part responsible for the tendency of temperament researchers to focus primarily on negative emotionality when studying infants (Fox & Stifter, 1989). Moreover, frequent expressions of negative affect in infancy are normative (Greenspan, 1991) and may serve as a marker for ANS integrity (e.g., Porges, 1996). This raises the possibility that RSA marks emotional competence in both infancy and toddlerhood and that the apparent shift in RSA–behavior relations from negative toward positive emotionality is an artifact of normative developmental shifts in emotional expression. Consistent with this interpretation, high negative reactivity scores of appropriately responsive infants are not predictive of negative emotionality later in life (see Gunnar, 1990). This observation may also stem from the types of tasks employed by researchers assessing emotionality in infant samples. Frequently, negative emotionality is assessed using somewhat intrusive challenges, such as the arm restraint procedure employed by Fox (1989; Stifter & Fox, 1990). Negative emotional expression in response to arm restraint may thus reflect adaptive reactivity and is not, in and of itself, predictive of subsequent negative outcomes, such as toddler noncompliance (Stifter, Spinrad, & Braungart–Rieker, 1999).

In addition, because positive affect differentiates before social behaviors, it is also possible that RSA–social competence relations are effected through emotionality. This inter-

pretation seems particularly likely given the previously mentioned findings by Eisenberg and colleagues (Eisenberg et al., 1995; Fabes et al., 1994) relating emotion regulation to social competence.

It has also been suggested that the age range from infancy to toddlerhood is a critical period for the development of emotion regulation, or the ability to modulate affective states internally. Calkins and Fox (1992; Calkins, 1994; Fox & Calkins, 1993) have proposed a model in which temperamental reactivity interacts with caretaking style to result in specific behavioral patterns. According to this model, two possibilities obtain for the emotionally and behaviorally reactive infant who exhibits high RSA. If the primary caretaker characteristically meets infant emotional reactivity with negative affect and intrusive control attempts, which tend to escalate rather than regulate arousal (e.g., Snyder, Edwards, McGraw, Kilgore, & Holton, 1994), behaviors and emotions will not differentiate, resulting in an aggressive toddler. Alternatively, if the primary caretaker is responsive and carefully modulates affective expressions through unintrusive deescalation, a highly social, outgoing toddler results. Note that this model implicitly predicts two groups with high RSA, one socially competent and appropriately expressive and the other aggressive and emotionally unregulated. The studies reviewed thus far, which were all conducted using normative samples, address only the former group. Support for the model might be suggested if aggressive samples were found to exhibit baseline RSA equivalent to that observed in socially competent children, or if children with high RSA who were exposed to coercive parenting were found to be aggressive. Because no studies have addressed this question in the toddler age range, the Calkins and Fox model has not been tested directly. In samples of older children, however, no such patterns are represented in the literature. Gottman and Katz (1995), for instance, found no relationship between externalizing behavior problems and exposure to marital hostility for children with high RSA. Marital hostility was a strong predictor of behavior problems, however, for low-RSA children. Thus, parental

hostility did not affect the high-RSA participants in the manner predicted by the Calkins and Fox model. Moreover, emergent evidence suggests that RSA may be reduced rather than elevated in behaviorally disordered children and adolescents. Such evidence was reported by Pine and colleagues (Pine, Wasserman, Coplan, Fried, Sloan, et al., 1996; Pine, Wasserman, Miller, Coplan, Bagiella, Kovelenuk, Myers, & Sloan, 1998), using a sample of the 6.5- to 10.5-year-old brothers of convicted delinquents. RSA was inversely related to scores on the externalizing scale of the Child Behavior Checklist (Achenbach, 1991), a finding also reported by Field, Lang, Martinez, Yando, Pickens, and Bendell (1996) in the children of dysphoric mothers. Similar results have been reported by Eisenberg and colleagues (1995), who found negative correlations between RSA and externalizing behavior problems in 6- to 8-year-old boys, a pattern also observed in adolescent and adult samples, to which this discussion now turns.

Adolescent and adult studies

In a study of 15-year-old adolescent males, Mezzacappa et al. (1997) reported reduced RSA in a group of 63 aggressive participants, compared to 59 controls. Aggression scores were derived from a variety of measures collected yearly via mother, teacher, and self-report, at ages 10–15 years. RSA was also related inversely to trait hostility in a sample of young adults (Sloan et al., 1994), as indexed by the Cook–Medley Hostility Scale (Cook & Medley, 1954). No additional studies addressing the relation between RSA and aggression have been reported. Thus, although the number of findings is not large, results from both children and adults suggest reduced parasympathetic tone in aggression.

These reports have come as a surprise to many researchers, who subscribed to the assumption that vagotonia (Eppinger & Hess, 1910/1915), or an autonomic imbalance favoring the PNS, was a marker of delinquent and aggressive behavior (see Venables, 1988). This assumption is rooted in two empirical relations that merit further discussion. First, at all age ranges considered, aggressive samples

are characterized by reduced resting heart rate relative to controls, a finding that has been widely replicated (e.g., Kindlon, Tremblay, Mezzacappa, Earls, Laurent, & Schaal, 1995; Raine, Venables, & Mednick, 1997; Rogness, Cepeda, Macedo, Fischer, & Harris, 1990; Wadsworth, 1976; see also Raine, 1993, for a review). Second, both within- and between-subjects comparisons suggest that heart rate is inversely related to RSA, as assessed across a variety of task conditions, and in response to pharmacologic blockade (e.g., Cacioppo, Uchino, & Berntson, 1994; Grossman et al., 1991; Grossman & Kollai, 1993; Grossman & Svebak, 1987; Hayano et al., 1991; see also Berntson, Cacioppo, & Quigley, 1995, for a review). Taken together, these findings suggest that aggression should be marked by *increased* RSA, which is contrary to the results reported above. However, only recently has the relation between heart rate and RSA been explored in aggressive groups. Thus, the vagotonia hypothesis is an extrapolation from heart rate-RSA relations in nonaggressive samples, a practice that has been questioned by developmental psychopathologists, as outlined in the introduction of this article.

Moreover, recall that both the sympathetic and the parasympathetic branches of the ANS contribute to patterns of cardiac activity. Because each branch can function somewhat independently of the other, inferences regarding sympathetic or parasympathetic activation based on heart rate alone are of questionable validity (see Berntson et al., 1994). This is because the activity of each branch can take on any number of values toward producing a given heart rate. Thus, concurrent sympathetic and parasympathetic dysregulation could result in both reduced heart rate and reduced RSA. Baseline sympathetic underarousal has been suggested as the cause of the low resting heart rates observed in aggressive samples (Raine, Venables, & Williams, 1990a; Raine, Venables, & Williams, 1990b). If combined with reduced parasympathetic tone, as suggested by the studies previously cited, sympathetic underarousal provides a potential explanation for the observed reductions in both heart rate and RSA in aggressive groups.

Consistent with this interpretation, Mezzacappa and colleagues (Mezzacappa, Tremblay, Kindlon, Saul, Arseneault, Pihl, & Earls, 1996; Mezzacappa et al., 1997), reported reduced heart rate and reduced RSA in antisocial adolescents, compared to controls.

The inverse relation between RSA and aggression is also surprising given similar patterns of parasympathetic functioning in both anxiety and depression. Although few such reports were available when Dalack and Roose (1990) suggested that elevated rates of cardiovascular disease in depressed patients were due to reduced vagal influence, several empirical reports have appeared in the literature since. However, because both tricyclic and tetracyclic antidepressants attenuate RSA (Mezzacappa, Steingard, Kindlon, Saul, & Earls, 1998; Yeragani, Pohl, Balon, Ramesh, Gritz, Weinberg, & Merlos, 1992; Jakobsen, Hauksson, & Vestergaard, 1984), and because studies of selective serotonin reuptake inhibitors suggest both increases (Tucker, Adamson, Miranda, Scarborough, Williams, Groff, & McLean, 1997) and decreases (Rissanen, Naukkarinen, Virkkunen, Rawlings, & Linoila, 1998) in parasympathetic functioning, this discussion will be restricted to the subset of reports in which depressed patients were medication free during RSA assessment.

As alluded to, these studies suggest that depressed patients exhibit reduced RSA. Light, Kothandapini, and Allen (1998), for instance, reported that mildly depressed women, as assessed by the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, & Erbaugh, 1961), exhibited significantly lower baseline RSA than nondepressed controls. Similar findings were reported by Rechlin, Weis, Spitzer, and Kaschka (1994) in patients meeting DSM-III-R criteria for major depression, melancholic type. Moreover, these patients exhibited lower RSA than a group diagnosed with reactive depression. This pattern suggests that the attenuated RSA in the former group may have resulted from enduring constitutional characteristics. Two additional studies have been reported in which melancholic and reactive subgroups were not separated. In each case, nonsignificant trends were reported, with depressed participants exhibit-

ing reduced baseline RSA compared to controls (Moser, Lehofer, Hoehn-Saric, McLeod, Hildebrandt, Seimbrenner, Voica, Liebmann, & Zapotoczky, 1998; Yeragani, Pohl, Balon, Ramesh, Glitz, Jung, & Sherwood, 1991). It is conceivable that significant effects were masked in these studies by including both depression subtypes in one group. Finally, two studies have reported significantly reduced RSA (Carney et al., 1995; Krittaya-phong, Cascio, Light, Sheffield, Golden, Finkel, Glekas, Koch, & Sheps, 1997), and one a trend toward reduced RSA (Carney, Rich, teVelde, Saini, Clark, & Freedland, 1988), in depressed versus nondepressed patients with coronary artery disease. In each case, 24-hr ambulatory ECG monitoring was employed. Thus, group differences reflect both RSA and RSA reactivity, which complicates interpretation. Nevertheless, these studies provide additional evidence of attenuated RSA in cases of depression.

Similar results obtain in anxiety-disordered patients. Once again, however, discussion must be restricted to reports in which medications were not employed, since benzodiazepines, which are commonly used to treat anxiety, alter vagal outflow (Adinoff, Mefford, Waxman, & Linnoila, 1992; Tulen et al., 1994). Lyonfields, Borkovec, and Thayer (1995) reported reduced RSA in patients who met DSM-III-R criteria for generalized anxiety disorder (GAD), a finding that they later replicated (Thayer et al., 1996). In the only other report addressing GAD in the literature, Kollai and Kollai (1992) found no significant RSA effect, although a trend was observed in the predicted direction. Similar differences have been found between control participants and patients with functional dyspepsia, a disorder characterized by chronic indigestion and epigastric discomfort with no definable organic cause (Haug et al., 1994; Hveem, Svebak, Hausken, & Berstad, 1998). Of note, patients with functional dyspepsia are characteristically anxious and often depressed (Langeluddecke, Goulston, & Tennant, 1990). Finally, reduced RSA has also been reported in anxious adolescent males (Mezzacappa et al., 1997).

A growing body of literature also suggests

that RSA is attenuated in patients who experience panic symptoms (see Friedman & Thayer, 1998a). Friedman et al. (1993), for example, reported significantly reduced RSA in a sample of female undergraduates who had experienced at least one panic attack in the past month, compared to a control group of blood phobics. This finding was later replicated with an expanded sample including male undergraduates and a normal control group (Friedman & Thayer, 1998b). Moreover, similar results have been reported in patients who meet DSM-III-R criteria for panic disorder (Yeragani, Pohl, Berger, Balon, Ramesh, Glitz, Srinivasan, & Weinberg, 1993). Of particular interest is the finding of Yeragani et al. (1991), who demonstrated significantly lower RSA in panic disorder patients than in *both* depressed patients and controls, suggesting more severely compromised parasympathetic functioning in the panic group.

Summary and implications

Two important points follow from this discussion. First, the empirical literature regarding RSA is most interpretable when viewed within a developmental context. RSA appears to mark the capacity for active engagement of infants with the environment, as reflected by temperamental reactivity, attentional capacity, and negative emotionality. In later infancy and toddlerhood, RSA marks measures of social competence and expressions of positive affect, including empathy. This apparent shift parallels normative developmental increases in RSA (Harper et al., 1978; Katona et al., 1980; Richards, 1985b), and age-appropriate changes in affective expression in the first years of life. Thus, at all age ranges considered, RSA indexes appropriate engagement and emotion regulation, which is manifested in older children as social competence. Consistent with this interpretation, girls, who are often more socially skilled and emotionally regulated than boys (e.g., Eisenberg et al., 1995, 1996; Fabes et al., 1993), exhibit higher RSA.

Second, studies of atypical samples suggest emotional deficits in cases of attenuated

RSA. A now sizable body of literature links reduced baseline vagal activity to depression, anxiety, and panic in adults. Although few reports speak to internalizing disorders in childhood, those that do suggest a similar pattern. Moreover, reduced RSA also characterizes aggressive adolescents and hostile adults. Although the behavioral dissimilarities among these disorders are extensive, all share the common feature of a dysregulated affective style. Aggression is characterized by anger and rage, depression by sadness and dysphoria, and anxiety by fear and panic. Thus, there appears to be a link between emotional inflexibility and baseline RSA, a topic that will be returned to in later sections of this article.

Vagal Reactivity, Emotion Regulation, and Attention

Infant studies

Because researchers were not yet aware that RSA and RSA reactivity mark somewhat different aspects of psychological functioning, early studies rarely included assessments of the latter index. Consequently, fewer reports of RSA reactivity are available for review. Moreover, few of the studies in which infant or child RSA reactivity has been reported have controlled for respiration rate, the importance of which has been outlined previously. Thus, reported changes in RSA in response to environmental demands may be confounded with changes in respiration rate and tidal volume. Reports of heart rate reactivity, which are more common, are also difficult to infer vagal reactivity from because of the aforementioned dual innervation of the heart by both the sympathetic and parasympathetic ANS branches. Due to these caveats, the extant research addressing RSA and heart rate reactivity in infants and children is somewhat difficult to interpret. Nevertheless, a review is presented below.

Although researchers assessing infant attention have generally not measured RSA reactivity directly, heart rate decelerations during visual stimulus presentations have been widely replicated (Casey & Richards, 1988; Lansink & Richards, 1997; Linnemeyer &

Porges, 1986; Richards, 1985a, 1987; Richards & Gibson, 1997). According to Porges and colleagues, these decreases in heart rate reflect activity of the “vagal brake,” which, according to polyvagal theory, moderates sympathetic output when attentional allocation is the appropriate coping strategy (see Porges et al., 1996). Thus, during periods of sustained attention requiring minimal physical or psychological effort, heart rate decelerations are presumed to be parasympathetically mediated. However, when RSA reactivity is measured directly during such tasks, the observed heart rate decreases are paralleled by concurrent *reductions* in RSA, which suggests decreased vagal influence. Such RSA reductions have been observed in infants (Richards & Casey, 1991), children (Weber et al., 1994), and adults (see Van der Molen, Bashore, Halliday, & Callaway, 1991). One interpretation of these findings, given the independent effects of respiratory parameters on vagal tone outlined previously (see Berntson et al., 1993, 1997), is that attention-related reductions in respiratory depth and tidal volume result in reduced RSA, despite increases in vagal efference. This interpretation is unlikely, however, given the findings of Richards and Casey (1991), who reported a negative correlation between heart rate and respiration during sustained attention in 14- to 26-week-old infants, but no relation between RSA and respiration. Thus, although RSA was suppressed during attention phases, the suppression could not have resulted from respiratory influences. Nevertheless, the authors attributed attention-related heart rate decelerations to increased vagal efference. The rationale for this interpretation is that RSA results from brain stem mechanisms, as previously outlined (see also Berntson et al., 1993), whereas heart rate reactivity during sustained attention is influenced by neocortical structures that directly inhibit vagal efferent traffic (see Richards & Casey, 1991). Thus, heart rate deceleration may be a more valid index of vagal reactivity than RSA during tasks requiring sustained attention.

To complicate interpretive efforts further, researchers have reported RSA reductions, accompanied by either no change in heart rate

(Huffman, Bryan, del Carmen, Pedersen, Doussard-Roosevelt, & Porges, 1998), or heart rate accelerations (e.g., Fracasso, Porges, Lamb, & Rosenberg, 1994), during more challenging tasks that require active engagement. Moreover, heart rate decelerations in the absence of RSA changes have also been reported (Porges et al., 1996). Thus, further studies are required toward clarifying the relation between RSA reactivity and environmental challenge in infants.

Regarding behavioral correlates of RSA reactivity during such tasks, Huffman et al. (1998) demonstrated that 3-month-old infants who responded to a laboratory-administered assessment of temperament with reduced RSA, were rated higher by their mothers on the soothability and the duration of orienting scales of the Infant Behavior Questionnaire than were infants who exhibited RSA increases. DeGangi, DePietro, Greenspan, and Porges (1991) compared baseline RSA and RSA reactivity between groups of normal and regulatory-disordered 8- to 11-month-old infants. The latter group, which was characterized by maternal reports of distractibility, irregular sleeping and feeding patterns, lengthy bouts of crying, and excessive irritability, did not differ from controls on baseline RSA. However, within-groups analyses identified an inverse relation in the dysregulated group between RSA and RSA reactivity during administration of the Bayley Scales of Infant Development (Bayley, 1969). Thus, contrary to the law of initial values, which suggests that participants with higher basal levels on a psychophysiological measure have a greater capacity for decreases (Benjamin, 1963), regulatory-disordered infants with high RSA exhibited the least RSA suppression during cognitive challenge, a pattern that was reversed for the control group. Finally, 9-month-old infants who exhibited relatively small reductions in RSA during administration of the Bayley Scales were rated by their mothers as more aggressive, more depressed, and more withdrawn on the Child Behavior Checklist at age 3 years than were infants who exhibited larger RSA reductions (Porges et al., 1996). Although both the DeGangi et al. (1991) and Porges et al. (1996) articles should be inter-

preted with caution due to small sample sizes, these studies, along with the Huffman et al. (1998) article, suggest that RSA reductions in response to challenge mark competent regulation. According to polyvagal theory, this RSA suppression reflects vagal withdrawal, which facilitates sympathetically mediated metabolic output, as reflected in heart rate increases, to cope efficiently with environmental demands (see Huffman et al., 1998). By this reasoning, the larger reductions in RSA reported by well-regulated infants reflect competent engagement with challenge.

In addition, most studies that have assessed both RSA and vagal reactivity have demonstrated significant relations between the two, with high baseline RSA predicting degree of vagal reactivity (Calkins, 1997; Porges et al., 1996; Porter et al. 1988; Richards, 1985a). This has been interpreted as indicating that high-RSA infants, who are behaviorally and emotionally reactive, are able to regulate their reactivity by appropriately allocating cognitive and motivational resources, as reflected in the application of the vagal brake. As mentioned previously, however, larger reactivity can be expected of those with higher baseline scores, and no statistical controls for baseline RSA were employed in any of the above reports. Moreover, respiratory frequency has been reported to vary inversely with task difficulty during attention demanding or cognitively challenging tasks (Denot-Ledunois, Vardon, Perruchet, & Gallego, 1998). Thus, further studies are required in which both respiration rate and baseline RSA are controlled before the vagal brake hypothesis can be confirmed.

Reports have also emerged suggesting that vagal reactivity reflects emotional responsiveness, as indexed by shifts in mood state. Bazhenova and Porges (1997), for instance, elicited RSA increases in 5-month-old infants by manipulating emotional state toward the positive, and RSA decreases by manipulating emotional state toward the negative. Thus, RSA was highest during a toy presentation condition, and lowest during a still-face condition in which the experimenter gazed unexpressively at the infant. Although RSA reductions in the latter condition could have

resulted from attentional processes, such an interpretation is unlikely for two reasons. First, the procedure has been associated with expressions of negative affect, and similar reductions in RSA elsewhere (Weinberg & Tronick, 1996). Second, the RSA reductions were accompanied by heart rate acceleration rather than deceleration, a pattern suggestive of vagal withdrawal, which is commonly observed in older participants while performing tasks that are psychologically stressful (Berntson, Cacioppo, Binkley, Uchino, Quigley, & Fieldstone, 1994; Cacioppo et al., 1994; Murphy, Alpert, Willey, & Somes, 1988; Kelsey, 1991). Regarding positive affect, DiPietro, Porges, and Uhly (1992), in a similar procedure, demonstrated that infants who reacted to a toy stimulus with phasic increases in RSA, engaged subsequently in more exploratory play.

Child studies

Three studies have reported relations between mood state and vagal reactivity in older children. Calkins (1997) subjected 2- and 3-year-olds to conditions designed to elicit both positive and negative affect. In the former condition, children were engaged in a puppet game, while in the latter they were prevented from playing with a musical toy that was placed in a clear plastic box. In both cases, RSA was suppressed from baseline. Once again, however, these results are difficult to interpret because attentional and respiratory processes might have contributed to the reductions in RSA. An alternative possibility, since the children were not prevented from moving, is that vagal withdrawal associated with metabolic output suppressed RSA. In the second study, Miller and Wood (1997) monitored RSA while asthmatic children watched a video containing both sad and happy scenes. Increases in RSA and decreases in heart rate were observed in response to both types of scenes, with maximal change during the sad presentations. These findings are inconsistent with reports from infant samples, and they are particularly difficult to interpret because asthmatic children are known to experience increased rates of affective disorders (e.g., Ben-

nett, 1994), which are characterized by vagal dysregulation in adults. In addition, excessive vagal influence from the dorsal motor nucleus (i.e., the vegetative vagus) has been suggested as a possible cause of asthma (Porges, 1995). Moreover, no control group was employed, and the only other report assessing vagal reactivity in response to mood induction produced a null result (Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996). Thus, further study is required before drawing any conclusions about mood state and vagal responding in this age group.

As with infant studies, a clearer relation exists between vagal reactivity and attention. When asked to count target tones presented within a string of distracters, 5- to 9-year-old boys exhibited reduced heart rate and reduced RSA from baseline (Weber et al., 1994). In addition, during more demanding tasks, reduced RSA and heart rate accelerations have been observed in both fourth- and fifth-graders (Suess et al., 1994), and 7- to 12-year-olds (Hickey, Suess, Newlin, Spurgeon, & Porges, 1995). These findings are consistent with reports from the infant literature linking heart rate and RSA reactivity to sustained attention and cognitive challenge.

Adolescent and adult studies

Although few studies have directly assessed the relation between vagal reactivity and sustained attention in adolescents or adults, the results of those that have are consistent with the infant and child literature. Porges and Raskin (1969), for example, reported reduced RSA and heart rate in college students during tasks requiring them to count both auditory and visual stimuli. Additionally, Coles (1972) reported heart rate deceleration during simple visual search tasks. As with infant and child studies, when psychological stress is imposed by increasing task difficulty (e.g., by requiring participants to perform serial subtraction), heart rate increases are observed, a finding that is well replicated (Berntson et al., 1994; Cacioppo et al., 1994; Murphy et al., 1988; Kelsey, 1991).

The bulk of the remaining studies in which vagal reactivity has been assessed have exam-

ined samples of depressed and anxiety-disordered adults. When group differences have been found, panic disorder patients have exhibited greater vagal reactivity than depressed patients, anxiety disordered patients, or controls. Yeragani et al. (1991), for example, examined vagal withdrawal in panic patients when moving from a supine to a standing position. Although such postural changes are associated with normative drops in vagal output, reductions were more profound in the panic group than in depressed or control groups, who did not significantly differ from one another. Moreover, no differences were found in posture-induced blood pressure changes, eliminating this as a potential mediator, via baroreceptor-induced parasympathetic withdrawal, of the group difference in vagal reactivity. In a second study using a similar procedure, significant differences in RSA were found between panic and control patients in both supine and standing positions (Yeragani et al., 1993). Although the groups exhibited comparable drops across conditions, RSA in the former group was consistently lower. Thus, contrary to the "law of initial values" (Benjamin, 1963), the proportion of RSA reduction, as a function of basal RSA, was larger in the panic patients. These findings suggest a link between excessive vagal withdrawal and panic attacks, an assertion that is supported by studies in which RSA was attenuated by hyperventilation and sodium lactate infusion, which are known to precipitate panic symptoms (e.g., Asmundson & Stein, 1994; George, 1989).

In addition, two studies have linked worrying, which characterizes both generalized anxiety and panic disorder, to vagal withdrawal. Thayer et al. (1996) subjected a group of patients who met DSM-III-R criteria for generalized anxiety disorder to a relaxation condition, and to a condition in which they were instructed to imagine a topic of greatest concern. Compared to control participants, generalized anxiety patients exhibited reduced RSA in both conditions. In addition, both groups demonstrated significant reductions in RSA from relaxation to worry. Similar findings have been reported by others (e.g., Lyonfields et al., 1995). Moreover, nonpharmacological

treatments that are effective in reducing anxiety augment RSA. Lehrer, Hochron, Mayne, Isenberg, Lasoski, Carlson, Gilchrist, and Porges (1997), for example, reported increased RSA in asthmatic adults during relaxation therapy. Although interpretational issues have already been noted with respect to asthmatic participants, similar increases in RSA have been reported in a variety of samples during hypoventilation (Asmundson & Stein, 1994; Yeragani et al., 1991, 1993), after prolonged exercise training (Al-Ani, Munir, White, Townsend, & Cooté, 1996; Shi, Stevens, Foresman, Stern, & Raven, 1995) and after massage therapy (Field, 1995). All of these procedures offer known anxiolytic effects. Thus, several sources of evidence suggest that vagal reactivity reflects intraindividual shifts in anxiety level, and that those with low baseline RSA are at particular risk for panic attacks during periods of acute vagal withdrawal.

Finally, Gottman and colleagues (Gottman, Jacobson, Rushe, Shortt, Babcock, La Taille, & Waltz, 1995; Jacobson, Gottman, & Shortt, 1995), reported vagal withdrawal during marital conflict in 80% of a sample of violent males. When considered in conjunction with the findings presented above regarding and panic attacks and anxiety, excessive vagal withdrawal may be a nonspecific marker of emotional lability.

Summary and implications

Although the research addressing RSA reactivity is less extensive and more difficult to interpret than the research addressing basal RSA, several patterns emerge, most of which should be interpreted with caution. Increased vagal efference, as inferred from heart rate deceleration, appears to characterize periods of sustained attention, a relation that has been observed at all age ranges considered. Although this finding is consistent with the polyvagal hypothesis that vagal inhibition subserves mammalian attentional processing, further research is required in which respiratory parameters and basal RSA are controlled before definitive conclusions are drawn. During more challenging tasks that require active

cognitive effort, competent engagement is reflected in partial vagal withdrawal, which presumably facilitates preparedness of the SNS to respond to upcoming demands, in anticipation of potential increases in metabolic output.

Also consistent with polyvagal theory, excessive vagal withdrawal appears to be related to emotional lability of a fight–flight nature. This is suggested by research with panic patients, who consistently exhibit vagal withdrawal that is disproportionate given their already low baseline RSA, and from the finding of reduced RSA during marital conflict in violent males. To the extent that large reductions in RSA are associated with adaptive responses to danger, those with reduced basal RSA may be at risk for such fight–flight responding in situations of psychological or cognitive challenge. Thus, the hypothesis set forth here is that moderate vagal withdrawal is associated with optimal engagement and preparedness to respond, whereas excessive vagal withdrawal is associated with emotional lability.

Relations between vagal reactivity and normative shifts in mood state are less clear, both because fewer studies have addressed the issue and because movement artifacts, uncontrolled respiratory parameters, and attentional processes could explain many of the results. Nevertheless, findings from these analyses, and from exercise, relaxation, and anxiety-induction studies, suggest that intraindividual fluctuations in vagal outflow reflect emotional state. When combined with the empirical findings previously reviewed regarding RSA, two broad conclusions are implied regarding psychopathology. First, as already suggested, baseline deficiencies in parasympathetic tone are related to negative emotional *traits*, including disorders of depression, anxiety, and aggression. Second, excessive vagal withdrawal is related to negative emotional *states*, most notably panic and anger.

Thus, the vagal system appears to reflect attentional and emotional processes. This is consistent with polyvagal theory, in which both structural and functional aspects of emotion are presumed to have evolved in conjunction with fight–flight responding and associated attentional processes in mammals. By

itself, however, polyvagal theory cannot account for the behavioral dissimilarities between disorders characterized by aggression and disorders characterized by anxiety and panic. Although both may represent excessive fight–flight responding related to vagal dysfunction, nothing in polyvagal theory suggests why anger and aggression predominate in some disorders, whereas anxiety and panic predominate in others. As outlined in earlier sections, these responses are presumed to be mediated by the SNS only when inhibitory parasympathetic influence is either chronically insufficient or suddenly removed. Thus, explanation of individual differences in characteristically angry versus anxious response sets requires a theory of SNS regulation. Such a theory has been described in detail by Gray (1982a, 1982b, 1987a, 1987b) and applied toward characterizing disorders of behavioral disinhibition in childhood by Quay (1988, 1993). A brief description of Gray's theory as applied to undersocialized aggressive conduct disorder and attention deficit hyperactivity disorder follows.

Gray's Motivational Theory

Gray (1982a, 1982b, 1987a, 1987b) proposed three functionally distinct yet interdependent brain systems that govern behavior: the fight–flight (F/F) system; the reward system, or behavioral activation system (BAS); and the punishment system, or behavioral inhibition system (BIS). The F/F system subserves both escape behaviors and defensive reactions under conditions of frustration, punishment, and pain. Gray was least elaborative regarding this system, although he suggested that brain circuits in the ventromedial hypothalamus, the central gray matter, and the amygdala mediate F/F responding. Activity within this pathway is determined in part by the emotional significance attributed to a stimulus, which, as previously outlined, is also reflected in vagal reactivity. Indeed, both the F/F system and the vagus nerve receive direct input from the dorsal motor nucleus (see Heimer, 1995), providing a structural link between vagal withdrawal and the F/F emotions panic and rage. Thus, nonspecific activity within this pathway ac-

companies F/F responding. The hypothesis presented here is that the form of such responding is primarily determined by Gray's other motivational systems, the BAS and the BIS, described below.

Initially referred to as the reward system, but later renamed by Fowles (1980), the BAS subserves appetitive motivational functions, governing both approach and active avoidance behaviors. Thus, the system is responsible for maximizing rewards (approach behavior) and for minimizing punishments in situations where behavioral responses are required (active avoidance). According to Gray (1987a), neural mediation of the BAS is rooted in the dopaminergic pathway that includes the ventral tegmental area, the nucleus accumbens, and the ventral striatum.

The BIS, on the other hand, subserves aversive motivational functions, controlling passive avoidance and extinction. Through the production of fear and anxiety, the system actively inhibits appetitive behaviors when aversive consequences are anticipated. Gray hypothesized that neural mediation of the BIS is rooted in both the serotonergic projections of the raphe nucleus and the noradrenergic projections of the locus ceruleus.

Psychophysiological evidence suggests that both motivational systems are effected through the sympathetic branch of the ANS. With respect to the BAS, support for this conjecture is derived from three observations. First, behavioral activation requires expenditures of energy, and the functional role of the SNS has been viewed traditionally as one of mobilizing resources to deal with environmental demands (e.g., Heimer, 1995). Second, increases in cardiac output, which are required of behavioral activation, are facilitated in part by sympathetically mediated changes in the contractile force of the left ventricle (see Sherwood, Allen, Fahrenberg, Kelsey, Lovallo, & van Doornen, 1990; Sherwood, Allen, Obrist, & Langer, 1986). Third, the reticular nuclei that control PNS influences on cardiac functioning are phylogenically new developments, with maximum differentiation in the mammalian brain stem (Porges, 1994). Thus, these structures evolved after the biobehavioral systems subserving approach and

avoidance motivation. Activity in the BAS is therefore likely to be reflected in SNS activity.

Behavioral inhibition, on the other hand, is reflected in electrodermal responding (Fowles, 1980, 1988), evidence for which is twofold. First, nonspecific skin conductance responses (SCRs) increase under threat of punishment (e.g., Katkin, 1965); second, well-controlled experiments suggest that such responses are unaffected by reward. Tranel (1983) reported increases in SCRs in participants exposed to feedback for incorrect responses during a continuous performance task, but not in participants who received a monetary reward for correct responses. Furthermore, when participants' monetary incentives are dependent on active responding, changes in skin conductance are reduced in comparison to those who are instructed to inhibit responding (Sosnowski, Nurzynska, & Polec, 1991). Thus, skin conductance changes are observed during punishment and passive avoidance, thereby reflecting BIS activity. Moreover, the eccrine sweat glands from which electrodermal activity is derived are enervated exclusively by cholinergic fibers of the SNS, with no parasympathetic input (see Fowles, 1986).

Finally, Gray suggested that the BAS and BIS are actively opposed to one another, with net output impacting upon behavior. When BAS functioning predominates, either approach or active avoidance results. When BIS functioning predominates, passive avoidance is likely. This active opposition has been central to theories of behavior disorders in childhood, including both undersocialized aggressive conduct disorder (UACD) and ADHD (Fowles, 1980, 1988; Quay, 1993; Rogness, Javors, & Pliszka 1992). A brief description of each disorder follows.

UACD and ADHD

Conduct disorder (CD) is currently defined as a behavioral pattern of persistent and repeated violations of social rules and the rights of others (American Psychiatric Association, 1994). Diagnostic features of CD include aggression toward people and animals, destruction of property, deceitfulness and theft, and serious

violations of social norms. Each of these features includes several specific criteria, with the endorsement of any three being sufficient for a formal diagnosis. Thus, none of the four features is defining, allowing a given case to meet diagnostic criteria without any aggressive symptoms. Much of the literature on CD and delinquency however, supports a distinction between CD cases low in aggression, and a subgroup characterized by heightened impulsivity, lack of empathy, failure to form loyal bonds with peers, and persistent use of instrumental aggression (see Quay, 1986). The third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III; American Psychiatric Association, 1980) reflected this distinction by including the UACD subtype.

Although subsequent versions of the DSM have subsumed all CD cases into an inclusive diagnostic category, the validity of the UACD distinction has been supported at several levels of analysis. Members of this subgroup are less competent socially (Henn, Bardwell, & Jenkins, 1980), exhibit delinquent characteristics across a greater variety of situations (see Moffitt, 1993) and are more likely to become antisocial adults (Huesmann, Eron, Lefkowitz, & Walder, 1984; Robins, 1966) than are their nonaggressive counterparts. In addition, twin studies suggest that the genetic loading for aggression is significantly higher than for otherwise delinquent but nonaggressive acts (Edelbrock, Rende, Plomin, & Thompson, 1995; Jary & Stewart, 1985). Finally, those meeting UACD criteria are more behaviorally disinhibited than other CD cases (Daugherty & Quay, 1991; Shapiro, Quay, Hogan, & Schwartz, 1988).

Behavioral disinhibition is also a core attribute of ADHD. Diagnostic features of ADHD include both inattention and hyperactivity–impulsivity (American Psychiatric Association, 1994). Each feature includes several specific criteria, with any combination of six being sufficient for a formal diagnosis. Because the criterion lists for both inattention and hyperactivity–impulsivity are lengthy (13 criteria each), neither feature is defining. Thus, diagnoses are subtyped as predominantly inattentive, predominantly hyperac-

tive–impulsive, or combined. Although those diagnosed with the latter two subtypes share behavioral disinhibition with UACD groups, they are not characteristically aggressive. Rather, their impulsivity is manifested in marked impatience, interruption of others, and difficulty regulating excitement.

Despite these differences, the behavioral disinhibition of both disorders is proposed to result from an imbalance in BAS and BIS functioning favoring behavioral activation. With few exceptions (e.g., Tremblay, Pihl, Vitaro, & Dobkin, 1994), this imbalance is theorized to result from an underactive BIS, which leads to impulsivity because the BAS operates relatively unopposed.

Consistent with this interpretation, aggressive children and adolescents exhibit diminished electrodermal activity at baseline (e.g., Delameter & Lahey, 1983), in response to orienting stimuli (e.g., Schmidt, Solanto, & Bridger, 1985), and in response to mental stressors (McBurnett, Harris, Swanson, Pfiffner, Freedland, & Tamm, 1993). Thus, UACD groups, who are deficient in behavioral inhibition, exhibit reductions in electrodermal activity across multiple paradigms.

Moreover, evidence at the neurotransmitter level for an underactive BIS in UACD is substantial. Kruesi, Rapoport, Hamburger, Hibbs, Potter, Lenane, and Brown (1990) reported a significant negative correlation between aggressive symptoms and concentrations of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the cerebrospinal fluid of 29 children, a finding that has also been reported in aggressive adults (Linnoila, Virkkunen, Scheinin, Nuutila, Rimon, & Goodwin 1983). Similarly, Birmaher, Stanley, Greenhill, Tomy, Gavrilescu, and Rabinovich (1990) reported an inverse correlation between aggression and available blood platelet binding sites for serotonin. Each of these findings suggests dysregulated serotonergic functioning, as predicted by the underactive BIS hypothesis. Additionally, noradrenergic functioning has been tied to impulsive aggression. In UACD boys, Rogeness and colleagues (Rogeness, Hernandez, Macedo, & Mitchell, 1982; Rogeness, Hernandez, Macedo, Suchakorn, & Hoppe, 1986; Rogeness, Maas, Javors, Macedo, Harris, &

Hoppe, 1988) have repeatedly demonstrated significant reductions in plasma dopamine-beta-hydroxylase, an enzymatic precursor of norepinephrine (NE), the neurotransmitter of the noradrenergic system. Similar results obtain in adult populations of impulsive aggressives (see Kavoussi, Armstead, & Coccaro, 1997). Thus, both serotonergic and noradrenergic regulation are related to impulsive aggression, findings consistent with Quay's (1993) argument of deficient BIS functioning in UACD.

Similar sources evidence also support the underactive BIS hypothesis for ADHD. In a study by Iaboni, Douglas, and Ditto (1997), ADHD children were rewarded with a monetary incentive for turning off a specified light (among five). Extinction trials then ensued in which no rewards were given. ADHD children exhibited significantly smaller changes from baseline in skin conductance during extinction than did controls, suggesting insensitivity to punishment and reduced BIS functioning.

Additionally, Rogeness et al. (1986) have reported an inverse correlation between dopamine-beta-hydroxylase levels and attention deficit symptoms. Relative to controls, ADHD groups also exhibit reduced urinary MHPG (Shekim, Dekirmenjian, Chapel, & Davis, 1982; Shekim, Sinclair, Glaser, Horwitz, Javaid, & Bylund, 1987; Yu-cun & Yu-feng, 1984), a metabolite reflective of NE activity in the central nervous system, the ANS, and the adrenal gland. Thus, ADHD samples may also be characterized by noradrenergic dysregulation.

In comparison, anxiety and depressive disorders are proposed to result from an imbalance in BAS and BIS functioning favoring behavioral inhibition. Research by Kagan et al. (1987), in which behaviorally inhibited children exhibited increased urinary NE metabolites, supports this assertion. Additionally, Rogeness et al. (1988) have reported positive associations between plasma dopamine-beta-hydroxylase levels, separation anxiety disorder, and depressive disorders in child inpatients. These results suggest increased NE, and thus heightened BIS functioning in anxiety and depression. Moreover, dysregulated

noradrenergic functioning has also been implicated in anxiety and depression in adults (Brawman-Mintzer & Lydiard, 1997; Lake, Pickar, Ziegler, Lipper, Slater, & Murphy, 1982; Leonard, 1997; Veith, Lewis, Linares, Barnes, Raskind, Villacres, Murburg, Ashleigh, Castillo, Peskind, Pascualy, & Halter, 1994).

To summarize, although both inhibited and disinhibited samples exhibit vagal deficits and are therefore not differentiated on parasympathetic functioning, the groups do exhibit alternative motivational profiles. Anxiety and depression are characterized by heightened BIS activity, whereas UACD and ADHD are characterized by reduced BIS functioning. Gray's theory does not account, however, for the behavioral differences *between* disinhibited subgroups. Both ADHD and UACD groups are impulsive, yet the elevated levels of aggression by the latter group are not well accounted for by attenuated BIS activity alone. As the above literature review suggests, however, aggressive samples are characterized by deficiencies in parasympathetic functioning.

An Integrated Model of Autonomic Functioning

Such findings suggest that the role of the ANS in these disorders might be clarified through analyses of complex interactions between the SNS and PNS branches. While compromised RSA and excessive vagal reactivity suggest emotional dysregulation, whether or not that dysregulation is manifested in aggression, anxiety, or depression, is reflected in characteristic sympathetic response patterns. Moreover, the SNS branch must be parsed into two motivational subsystems, the BIS and the BAS, to account fully for behavioral differences across these disorders. This integrated model of ANS functioning is presented in Figure 2. Motivational predispositions, as reflected in BAS and the BIS functioning, fall under SNS control. Regulatory functioning, as reflected in vagal tone and vagal reactivity, falls under PNS control. Note that both ANS branches contribute independently to behavioral and emotional predispositions.

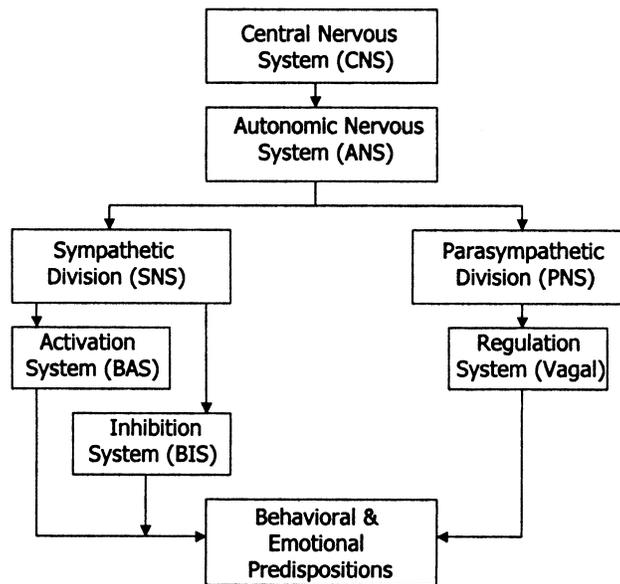


Figure 2. An integrated model of autonomic nervous system-behavior relations in which motivational functioning falls under sympathetic nervous system control and regulational functioning falls under parasympathetic nervous system control. Note that both branches contribute to behavioral predispositions.

Specific patterns of autonomic activity for the disorders addressed in this paper are summarized in Table 1. Heightened BAS activity characterizes appetitive behavioral tendencies, including those exhibited in extraversion, aggression, impulsivity, and panic, the latter of which is dominated by active avoidance. Heightened BIS activity characterizes passive and fearful behavioral tendencies, including those exhibited in introversion, depression, anxiety, and, once again, panic. Note that the clusters of behaviors related to BAS and BIS dysregulation map almost directly onto the novelty-seeking and harm-avoidance dimensions, respectively, of Cloninger's personality typology (Cloninger, 1987; Cloninger, Svrakic, & Przybeck, 1993; Cloninger, Svrakic, & Svrakic, 1997), and are thus rooted in validated patterns of behavioral functioning.

Regarding the PNS, low RSA characterizes dysregulated emotional states, including anger, depression, and anxiety. Excessive vagal reactivity, on the other hand, characterizes emotional lability, as reflected in panic attacks and impulsive aggression. According to this model, outcome cannot be predicted by the activity or reactivity in any single system.

Both the motivational component, contributed by the BIS and the BAS, and the regulational component, contributed by the vagal system, are required.

Although most of the cells presented in Table 1, particularly those related to vagal tone, vagal reactivity, and BIS functioning, are supported by the literature review presented, those related to BAS functioning are more speculative. Evidence reviewed previously, for example, suggests that both anxiety and depression are characterized by heightened BIS functioning and reduced vagal tone, yet little is known about BAS activity in either disorder. Similarly, while aggressive participants exhibit reduced BIS activity, reduced RSA, and heightened RSA reactivity, little direct evidence exists regarding BAS activity in disorders of disinhibition. Moreover, two schools of thought are represented in the literature. As suggested previously, one proposes that aggression results from reward dominance, or an overactive BAS (Fowles, 1980, 1988; Milich, Hartung, Martin, & Haigler, 1994; Quay, 1988, 1993). Others, however, have suggested that trait aggression represents a form of sensation seeking that results from

Table 1. Patterns of autonomic nervous system functioning in common psychopathologies and personality types

Motivational Predisposition	Behavioral Manifestation	Motivational System (SNS)		Regulatory System (PNS)	
		Activation (BAS)	Inhibition (BIS)	Emotional Trait (RSA)	Emotional State (RSA Reactivity)
Disinhibition	Impulsivity (ADHD)	High	Low	—	—
	Aggression (UACD)	High	Low	Low	High
	Panic	High	High	Low	High
	Extraversion	High	—	—	—
Inhibition	Anxiety	—	High	Low	—
	Depression	Low	High	Low	—
	Panic	High	High	Low	High
	Introversion	—	High	—	—
None	Emotional stability	—	—	High	—
	Emotional lability	—	—	—	High

Note: High, atypically high activity; Low, atypically low activity. Dashes represent normal activity. Entries in the BIS, RSA, and RSA reactivity columns are supported by the literature reviewed herein. Entries in the BAS column are more speculative and require empirical confirmation.

insensitivity to reward, and by implication an *underactive* BAS. According to this account, aggressive probands require larger rewards to experience hedonic states equivalent to those of controls. Thus, in the absence of sufficient inhibitory mechanisms (contributed by the BIS) these probands engage in instrumental aggression to attain satisfactory reward states. It should be noted that such stimulation-seeking formulations have a long history in the personality and aggression literatures (see Eysenck & Gudjonsson, 1989; Quay, 1965; Raine, 1993, 1996; Zuckerman & Como, 1983), and that similar accounts have been offered for ADHD (Haenlein & Caul, 1987). These alternative interpretations can be resolved only through direct assessments of BAS functioning, which to date have not been conducted.

Note also that activity levels within the BAS, BIS, and vagal systems are assumed to reflect continuously distributed individual differences, which may help to account for observed patterns of comorbidity within internalizing and externalizing disorder domains. Both ADHD and UACD, for instance, are proposed to be characterized by dysregulated BAS and attenuated BIS functioning. The latter disorder is also characterized by low RSA

and high RSA reactivity. Because those with ADHD are impulsive due to their BAS/BIS motivational profile, even typical degrees of vagal reactivity may place them at risk for aggressive behavior, suggesting that CD symptoms should be common in ADHD groups. Prevalence figures from both epidemiological and clinical samples support this supposition, with 30–50% of ADHD cases exhibiting comorbid CD (Moffitt, 1990; Quay, 1988; Sandberg, Weiselberg, & Shaffer, 1980). Moreover, because UACD shares each autonomic feature of ADHD, those with UACD should exhibit ADHD symptoms almost without exception, a prediction that is also borne out empirically (e.g., Klein, Abikoff, Klass, Ganeles, Seese, & Pollack, 1997). Similar reasoning suggests a potential explanation for the high rates of comorbidity observed among anxiety and depressive disorders (Maser & Cloninger, 1990), where differences along a continuum of BAS activity are the only discriminating autonomic feature in an otherwise identical profile.

Patterns of comorbidity observed across internalizing and externalizing disorders present a more difficult problem. The overlap between CD and depression, for example, has been estimated at as high as 35% in adoles-

cents (Kovacs, Paulauskas, Gatsonis, & Richards, 1988). Yet the only common feature that has been empirically documented across these disorders is low vagal tone. There are several potential explanations for this dilemma. Evidence suggests that BAS dysregulation in externalizing disorders may be specific to situations in which external incentives are present (see Milich et al., 1994). Thus, while conduct disordered participants are able to inhibit ongoing behaviors when those behaviors are not paired with external reinforcers (Schachar & Logan, 1990), they are not able to do so when cues for reward are present (Daugherty & Quay, 1991; Shapiro et al., 1988). Consistent with these findings, the proactive aggression characteristic of CD is not engaged in indiscriminately; it is often motivated toward instrumental rewards such as valued objects or social dominance (see Bandura, 1983; Dodge, 1991). In contrast, dysregulated BIS functioning may not be situation specific. As previously reviewed, UACD and ADHD participants exhibit reduced electrodermal responding across multiple paradigms (e.g., Delameter & Lahey, 1983; McBurnett et al., 1993; Schmidt et al., 1985). In addition, reports of reduced serotonin metabolites (Birmaher et al., 1990; Linnola et al., 1983) and NE precursors (Rogness et al., 1982, 1986, 1988) in aggressive samples also suggest chronic BIS dysregulation. These findings raise the possibility that concurrent BAS and BIS dysregulation could result in ongoing depression, punctuated by episodic aggressive outbursts when instrumental gains are accessible.

Finally, all depressive symptoms are probably not rooted in autonomic dysregulation. Aggressive behaviors exact many consequences for conduct disordered individuals, including damage to interpersonal relationships, loss of access to resources, and institutionalization. For those with some degree of insight, such consequences may be sufficient to precipitate depressive symptoms of an exogenous nature.

Psychophysiological Assessment of ANS Patterns

Clarifying many of these ambiguities depends upon the simultaneous assessment of multiple

autonomic systems and on more direct assessment of BAS functioning, two practices that to date have not been represented in the developmental psychopathology literature. To a large degree, this is probably due to the lack of an integrative theory of ANS functioning in psychopathology. In addition, findings of heart rate changes in response to incentive motivation, which have been proposed to index BAS functioning (Fowles, 1980, 1988), are difficult to interpret because heart rate is not a valid index of sympathetic activity, since both branches of the ANS contribute to chronotropic cardiac regulation (see Berntson et al., 1994). Thus, estimates of group differences in BAS activity that are based on heart rate are systematically confounded with differences in parasympathetic outflow. Although estimates of vagal influence are obtainable through the methods previously described, until recently limitations in technology have precluded noninvasive assessment of purely sympathetic effects on the heart. By using impedance cardiography, however, these limitations may be overcome. This technique enables researchers to assess cardiac preejection period (PEP), or the time between left ventricular depolarization and ejection into the aorta (see Sherwood et al., 1990). This interval is determined by beta-adrenergic, and thus sympathetic, influences (McCubbin, Richardson, Langer, Kizer, & Obrist, 1983; Mezzacappa, Kelsey, & Katkin, in press), with shorter intervals indicating greater sympathetic activity. Moreover, the equipment employed is noninvasive, which is also the case for assessments of both skin conductance and RSA. Thus, the exploration of complex sympathetic and vagal interactions through concurrent assessment of BIS, BAS, and vagal reactivity is now possible. Doing so is of course essential toward validating the proposed autonomic model.

We are currently conducting such an assessment with UACD, ADHD, and control groups of adolescent males. PEP, skin conductance, and RSA are being monitored during baseline, reward, extinction, and passive coping with threat. As previously suggested, the reward condition is expected to elicit BAS reactivity, as indexed by PEP, whereas the ex-

tion condition is expected to elicit BIS reactivity, as indexed by skin conductance responses. The passive coping task, in which participants view a videotape of an escalating conflict between peers, will be informative with respect to BAS, BIS, and vagal reactivity to social stimuli, which are more ecologically valid than incentive reward tasks. Both the UACD and ADHD subjects are expected to be differentiated from controls on electrodermal reactivity. Additionally, the UACD subjects are expected to be differentiated from ADHD subjects on PEP and RSA. The hypothesized pattern of results would support the need for concurrent assessment of both autonomic branches in characterizing behaviorally disinhibited groups. Subsequent studies are planned that will involve similar assessments with anxious and depressed samples.

Concluding Remarks

At the outset of this paper, it was suggested that several apparent discrepancies in the vagal tone literature could be accounted for by adopting three broad tenets set forth by developmental psychopathologists. The first of these tenets underscores the importance of studying development in our efforts toward understanding behavior. The consistent finding that RSA predicts behavioral reactivity and negative emotionality in infancy, but positive emotionality and social competence in later childhood, is no longer perplexing when linked to normative emotional development and differentiation. At all age ranges considered, vagal tone marks competent emotional expression and active engagement with the environment. Such engagement is tied to negative emotionality in infancy because negative affect dominates the normative emotional repertoire at that age. Later, vagal tone marks positive affect and emotion regulation capabilities, following normative shifts in social and emotional development.

The importance of the second tenet, that typical and atypical development must be juxtaposed toward fully understanding behavioral disorders, is illustrated by the unexpected finding that RSA is lower in aggres-

sive groups. Many researchers assumed that the index would be elevated in aggressive participants, because of (a) the positive association between RSA and negative emotionality in infancy, (b) reports of reduced RSA in cases of anxiety and depression, and (c) the well-replicated finding of reduced heart rate in aggressive populations. As reviewed, however, direct comparisons between controls and aggressive samples suggest that reduced RSA characterizes anger as well as anxiety, and is thus a nonspecific marker of dysregulated emotion.

This brings us to the final tenet, that behavior is multiply determined by complex interactions among biological and psychological systems. In this paper, the biological component was addressed through the advancement of an integrated theory of ANS functioning in psychopathology. Considerable evidence was presented outlining the importance of assessing separate motivational systems governing approach behaviors (the BAS), avoidance behaviors (the BIS), and emotional regulation (the vagal system). It was further suggested that complex interactions among these systems must be assessed, because in isolation none can sufficiently account for the diversity of findings reviewed.

Equally important, however, is to emphasize that although development must be considered toward explaining the large set of vagal tone-behavior associations presented, this is not a theory of vagal or emotional development. Because the current literature represents a set of correlational associations, causal inferences regarding the role of vagal tone in the development of emotion regulation, or regarding the role of socialization processes in the development of vagal tone and vagal responding, are not possible. The theory of emotional and behavioral differentiation offered by Calkins and Fox (1992; Calkins, 1994; Fox & Calkins, 1993), which represents the only attempt to address the role of socialization in PNS development and associated emotion regulation capacities, has not received empirical support. Furthermore, few of the substantial number of articles reviewed directly address issues of development through longitudinal research designs. Thus, although

vagal tone marks emotional competence at all ages, these relations are currently descriptive and support no causal interpretations.

In addition, our future understanding of the role of the ANS in behavioral and emotional functioning will be greatly enhanced if standardized methods of assessment are adopted by researchers in this area. As indicated in several sections of this paper, psychophysiological indices of vagal functioning are difficult to interpret without controlling for respiration rate, attentional load, and movement artifacts, which have been largely ignored in the infant and child literature to date. Our understanding of ANS functioning in psychopathology will be further enhanced when direct assessments of both sympathetic and parasympathetic influences on cardiac functioning are employed. The practice of inferring the activity of either autonomic branch from heart rate alone is outdated and unlikely to be fruitful in the future.

Finally, it should be noted that this theory is not intended to be a comprehensive account of personality, nor is this article intended to suggest that autonomically influenced behaviors are immutable. As Cloninger et al. (1997) have noted, personality is comprised of both temperamental traits, which this paper addresses, and character traits, which it does not. Although Cloninger has suggested that we are

born with the former and develop the latter, the position taken here is more agnostic. The extent to which genetic and environmental factors determine autonomic output is likely to involve complex interactions (Cicchetti & Tucker, 1994). Moreover, the current data are open to interpretation. Field et al. (1995), for example, have reported emerging differences in RSA across the 1st year of life between children of depressed and nondepressed mothers. Although the authors leaned toward an environmental explanation, differential developmental trajectories can be genetically determined, as was also acknowledged. It is possible and perhaps likely that genes determine a range of potential autonomic functioning, within which transactions between the developing organism and the environment contribute to observed levels of functioning. Parent-child relationship characteristics are thought to impact other genetically influenced biological systems in such a manner (e.g., Pine, Wasserman, Coplan, Fried, Huang, et al., 1996). Thus, further research is required to address the extent to which autonomic functioning is malleable. Such research may have important implications for future intervention efforts addressing impulsive and otherwise dysregulated behaviors. It is hoped that in the future these efforts and others will include concurrent assessments of multiple autonomic systems.

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