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Anxiety, Antisocial Behavior, and Heart Rate Regulation in Adolescent Males

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We explored relationships between anxiety and antisocial behavior and autonomic heart rate regulation in a homogenous sample ($N = 175$) of 15-year-old males. Measures of anxiety and antisocial behavior were obtained at yearly intervals over a period of 4–6 years. Components of heart rate variability associated with postural (sympathetic) and respiratory (vagal) change and transfer of respiratory to heart rate variability were estimated at age 15 using spectral analytic techniques. Anxiety and antisocial behavior were predictably related to enhanced and diminished levels of mean heart rate, respectively. Anxiety was also predictably related to enhanced sympathetic mediation of phasic postural effects on heart rate. Antisocial behavior was unexpectedly related to disruption of vagally mediated, phasic respiratory effects on heart rate. Anxiety and antisocial behavior showed distinct relationships to heart rate, and to the autonomically mediated components of heart rate variability from postural and respiratory sources. Spectral analytic techniques helped elucidate these unique regulatory patterns, suggesting utility for future research in this area.

Keywords: Antisocial behavior, anxiety, heart rate regulation.

Abbreviations: AHA: American Heart Association; POST power: postural source; RSA power: respiratory sinus arrythmia.

Introduction

Heart rate and heart rate variability have been employed in psychophysiologic research as noninvasive indices of autonomic nervous system regulatory function. In childhood and adolescence, heart rate–behavior relationships have been examined in a variety of circumstances. Investigators have identified relationships between various disruptive behaviors on the one hand and resting heart rate on the other. These findings indicate that heart rate is lower in conduct disorder, and in aggressive subjects (Kindlon et al., 1995; Raine, 1988; Raine & Jones, 1987). Furthermore, lower heart rate in pre- and mid-adolescence was found to be predictive of criminality in young adulthood (Raine, Venables, & Williams, 1990a, b; Wadsworth, 1976), whereas higher heart rate among antisocial adolescents was predictive of desistence of such behaviors in young adulthood (Raine, Venables, & Williams, 1994). Interestingly, these heart rate–behavior relationships have not been found to persist into adulthood (Hare, 1982; Raine, 1988), suggesting possible developmental "windows" for such phenomena (Venables, 1987), though comparisons in adults have been limited to psychopathic and nonpsychopathic criminals, thereby limiting the generalizability of the findings.

In contrast to the findings in pathologically disinhibited subjects, Kagan and colleagues (Kagan, Reznick, & Snidman, 1987, 1988) found that children characterized by behavioral inhibition and shyness demonstrated higher heart rate than their (nonpathological) less in-

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hibited counterparts. Garralda, Connell, and Taylor (1991), found that children with emotional disturbances demonstrated hyper-responsiveness of heart rate to aversive consequences during mental tasks, when compared with conduct-disordered children and nonclinical controls.

In one of the few examples where heart rate–behavior relationships in children were explored using spectral analytic methods to assess the heart rate variability, Kagan et al. (1987, 1988) noted that behaviorally inhibited children with higher heart rates, when exposed to the stress of cognitive tasks, showed greater shifts in the distribution of the heart rate variability towards lower-frequency regions of the power spectrum, where sympathetic influences are active. These findings support the notion that behavioral inhibition may be associated with enhanced sympathetically mediated effects on heart rate.

Related work with undersocialized conduct disorder led Quay (1993) to suggest that such subjects are less sensitive to cues for punishment, that is, less anxious or inhibited, in the presence of cues for rewards. In this model, conduct disorder is presumably related to lower anxiety, reduced noradrenergic function, and, by implication, lower heart rate. This model is empirically supported by studies showing lower levels of noradrenergic function in undersocialized aggressive conduct disorder (Rogeness, Javors, Maas, & Macedo, 1990; Rogeness et al., 1988). Complementary hypotheses involving autonomic under-arousal (Raine et al., 1990a, b), understood as reduced sympathetic (noradrenergic) influences, and passive vagal coping (Raine & Jones, 1987; Raine & Venables, 1984), have both been advanced as plausible explanations for the heart rate findings in behavioral disinhibition as well.

Taken together, then, these studies suggest that there are biological factors, which are manifest at one level as individual differences in heart rate, and implicitly in autonomic nervous system regulatory functions, that are associated with the development of either antisocial behavior or anxiety. However, the heart is dually innervated by the autonomic nervous system, and the sympathetic and parasympathetic branches have opposing actions on heart rate. As a result, reductions in heart rate, which might be construed as evidence of under-arousal, can in fact arise from enhanced vagal modulation, or from reduced sympathetic modulation. Similarly, increases in heart rate can arise either from heightened sympathetic or diminished vagally mediated influences. Furthermore, since individuals can show different physiologic response patterns depending on the experimental conditions or physiologic indices measured (Garralda et al., 1991; Zahn & Kruesi, 1993), the interpretation of observed response patterns must also take these contextual factors into account. Methods that can decipher the relative contributions to heart rate regulation of the two autonomic branches are increasingly important, then, when questions are raised about how different forms of psychopathology and the autonomic regulation of heart rate are related.

In an earlier study involving the present sample, Tremblay, Pihl, Vitaro, and Dobkin (1994) demonstrated a predictive relationship between teacher's behavioral assessments in kindergarten and early onset of antisocial behavior in adolescence. This study highlighted how early characteristics of behavioral disinhibition can persist to become frank antisocial behaviors later in development.

In the current study, we examined the relationship of such antisocial behavior and, in contrast, anxiety to heart rate and to the heart rate variability generated from postural and respiratory sources, which our experimental protocol was specifically designed to elicit. We quantified both heart rate and respiratory activity during the physiologic assessments as continuous time series.

We attempted to replicate the heart rate–behavior relationships of previous investigators in this homogenous sample of 15-year-old boys. We then determined if persistently high levels of antisocial behavior were associated with evidence for increased vagal mediation of respiratory-driven heart rate variability; and whether persistently high levels of anxiety were associated with evidence for increased sympathetic mediation of heart rate variability driven by postural change. Finally, we explored the relationships of anxiety and antisocial behavior to the central autonomic processing of the transfer of respiratory variability to heart rate variability.

Under the conditions of our protocol, it was expected that linear relationships would exist between the respiratory and the heart rate variability (Berger, Saul, & Cohen, 1989; Saul, Berger, Albrecht, Stein, Chen, & Cohen, 1991; Saul, Berger, Chen, & Cohen, 1989). It was also assumed that in the absence of respiratory pathology, cardiac pathology affecting the sinus node and associated conduction tissue, or neuropathy affecting the central or peripheral autonomic nervous systems, that any differences in the transfer of respiratory to heart rate variability would reflect differences in the central autonomic processing of the respiratory–heart rate relationship.

Methods

Subjects

Subjects were 175 15-year-old boys, participating in a larger longitudinal study in the city of Montreal, Canada, which began in 1984 when the boys were 6 years old (Tremblay et al., 1994). All subjects from the larger study ($N = 1037$) lived in the inner city, attended public schools, and were offspring of Canadian-born, French-speaking, Caucasian parents, with less than 15 years of education. Families of origin were all from the lower two of six socioeconomic status categories for Canadians, according to Blishen, Carroll, and Moore (1987), whose scale utilizes income, educational level, and occupational prestige to classify status. Our subsample, which has received more extensive laboratory assessments throughout the study, included an overrepresentation of boys who were rated as disruptive in kindergarten by teacher report on the Social Behavior Questionnaire (Kindlon et al., 1995; Tremblay et al., 1991, 1994).

Our subsample represented a group of boys with minimal absence of behavioral data over the time period of the study. Purposely included were a number of boys with early onset stable aggression ($N = 63$), and with stable absence of aggression ($N = 59$). A detailed description of how this sample was derived can be found in Seguin, Pihl, Harden, Tremblay, and Boulerice (1995).

Assessment of Anxiety and Antisocial Behavior

At ages 10, 11, 12, 13, 14, and 15, boys completed self-report questionnaires pertaining to the previous 12 months, except at age 10 when the time frame was "ever before." Scales for anxiety and antisocial behavior were extracted from these reports.

For antisocial behaviors, data were available at all ages. Items determined the extent to which subjects used aggression or the threat of aggression to obtain material gains or social domination, and to what extent other antisocial behaviors such as theft, vandalism, and use of drugs and alcohol occurred (Appendix 1) (Leblanc & Frechette, 1989; Tremblay et al., 1991, 1994).

For anxiety, data based on the Jesness Inventory of Adolescent Personality were available at ages 10, 11, 12, and 14. The items corresponding to the construct "Social Anxiety" determined the extent to which subjects experienced anxiety in relation to novelty, uncertainty, taking risks, social embarrassment, and social rejection (Appendix II) (Jesness, 1971, 1983, 1986; Jesness & Wedge, 1984, 1985; Martin, 1981).

Data from mother and teacher reports on the Social Behavior Questionnaire were available at 6, 9, 10, 11, 12, 13, 14, and 15 years of age. From these, scales for aggression and anxiety were extracted (Appendix III) (Kindlon et al., 1995; Tremblay et al., 1991).

Assessment of Height, Weight, Pubertal Status, and Physical Conditioning

Height, weight, and pubertal status (Petersen, Crockett, Richards, & Boxer, 1988) were measured in the laboratory. Boys were asked if they had played sports regularly during the previous year. If so, they named the three sports they played the most, and the approximate number of hours per week they devoted to each sport. The sports nominated were typically seasonal, so the mean number of hours per week for the three sports was used as an estimate of physical conditioning for the previous year. No attempt was made to weight the sports by degree of exertion.

Spectral Analysis of Heart Rate Variability

Beat-to-beat changes in heart rate are driven by variations in other processes such as respiration and blood pressure regulation. The sympathetic and parasympathetic branches of the autonomic nervous system mediate the preponderance of relationships between such sources of heart rate variability and the heart rate variability itself.

Spectral analysis of this sequential cardiac activity utilizes estimation techniques based on Fourier transformations (Brigham, 1974), which operate on time series information much like the effects of a prism on colorless light, separating the signal into component frequencies or colors, each component contributing to the total variance or power in the signal. Spectral analysis of cardiac time series portrays the variance in heart rate as a function of frequency. The area under the spectrum is the total variance or power in the signal over the frequency range studied (Cook & Miller, 1992). The distribution of that variance is associated with the particular sources of the heart rate variability and their corresponding parasympathetic and sympathetic nervous system mediators (Akselrod et al., 1981, 1985; Pomeranz et al., 1985).

Under ordinary circumstances, the preponderance of the heart rate variability is contained within the frequency range from 0.0 to 0.5 hertz (Hz). The power spectrum for this

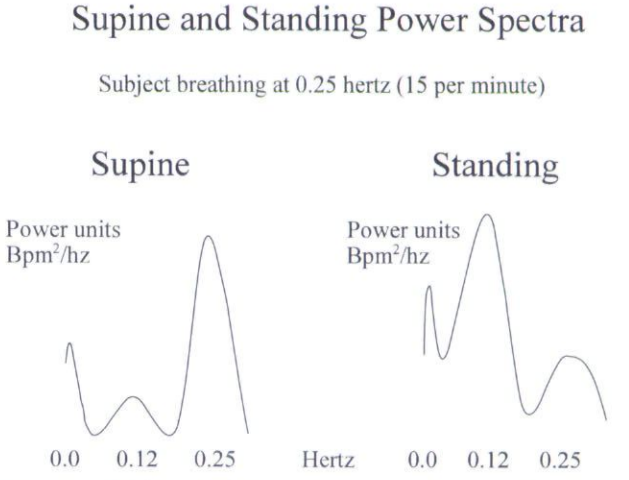

Figure 1. Depiction of typical power spectra resulting from short-term monitoring of heart rate variability. Note the change in the distribution of the heart rate variability due to posture change.

frequency range is commonly divided into low-frequency heart rate variability or power (below 0.04 Hz), mid-frequency power (between 0.04 and 0.15 Hz), and high-frequency power (above 0.15 Hz) (Fig. 1: Akselrod et al., 1981, 1985; Parati, Saul, DiRienzo, & Mancia, 1995; Pomeranz et al., 1985). In addition to autonomic influences, low-frequency power contains substantial contributions from thermoregulatory and metabolic influences on heart rate variability. Mid-frequency power is primarily driven by variations in blood pressure. In the standing posture, sympathetically mediated effects predominate in this region and vagal effects are inhibited, whereas in the supine posture vagally mediated effects predominate. High-frequency variability is primarily respiratory in origin, and vagally mediated (Berger et al., 1989; Saul et al., 1989, 1991). The spectral regions of interest in our experiment were the mid- and high-frequency bands.

When the sources of heart rate variability are quantified as time series concomitantly with the heart rate variability, aspects of the transfer of this variability can be estimated (Fig. 2). Using spectral techniques, this transfer is portrayed in the magnitude of the heart rate response to quantified changes in the source (gain or transfer magnitude) of the heart rate variability. For respiration, transfer magnitude is measured in beats per minute change in heart rate, per liter of lung volume change (bpm/liter), whereas heart rate change related to blood pressure regulation is measured in beats per minute of heart rate change per millimeter of mercury change in blood pressure (bpm/mm Hg). The timing of the heart rate responses to changes in the source (transfer phase in degrees), as well as the coherence (linearity and statistical reliability) of the relationship between the source and the heart rate response, can be estimated similarly (Fig. 3) (Berger et al., 1989; Saul et al., 1989, 1991). More detailed summaries of these spectral estimation techniques and their utility can be found elsewhere (Appel, Berger, Saul, Smith, & Cohen, 1989; Mezzacappa, Kindlon, Saul, & Earls, 1994; Saul, 1990).

Measurement of Heart Rate and Respiration

Physiologic assessments took place in a laboratory setting familiar to the subjects. A surface electrocardiogram (ECG) (lead II configuration) was obtained at 14 and 15 years of age, each time on the same day that the self reports were completed.

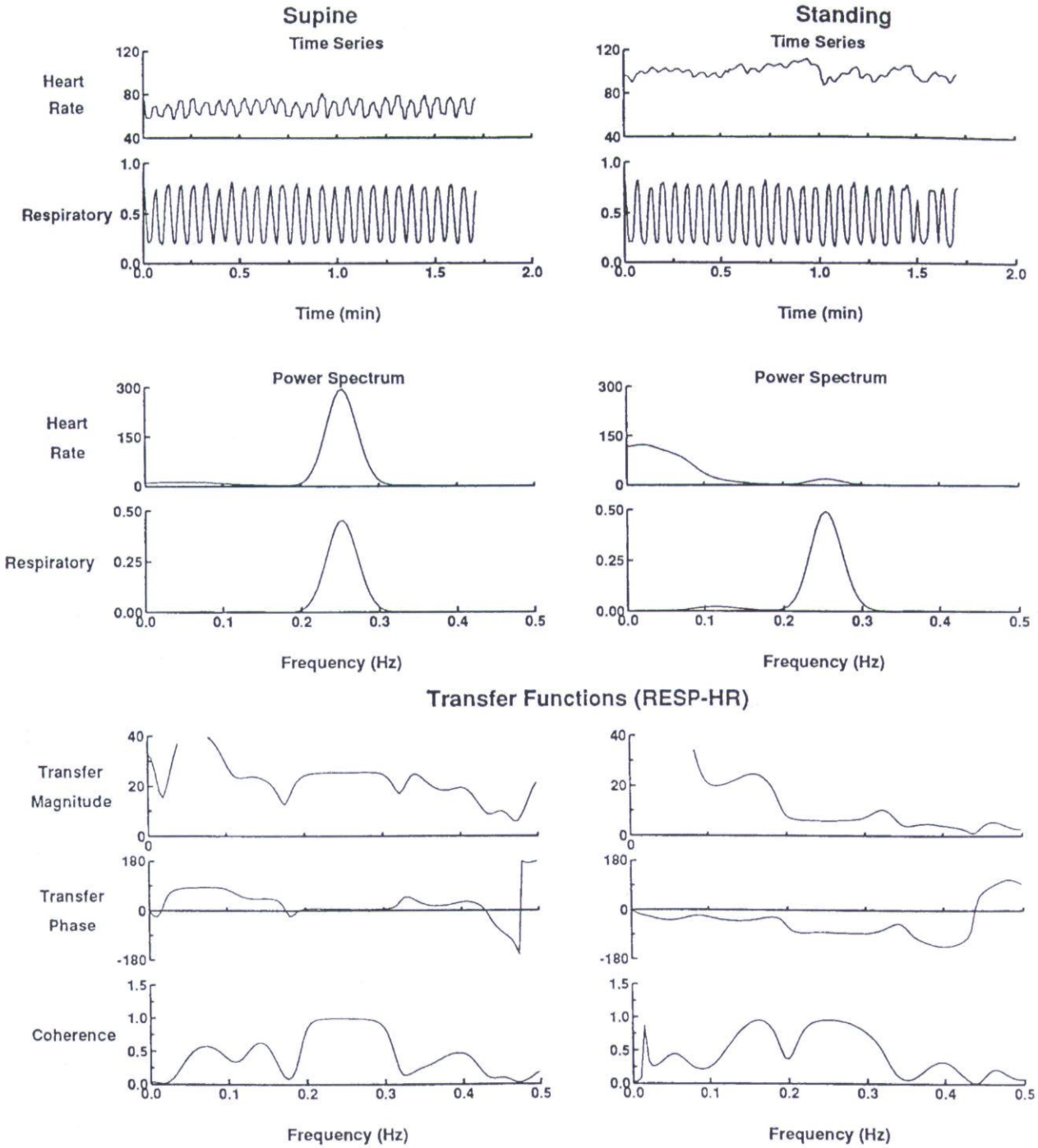

Figure 2. Respiratory and heart rate time series, power spectra, and transfer functions in a subject breathing at 0.25 Hz, in both the supine and standing postures. The region of primary interest for the transfer function estimates is that from 0.20 to 0.30 Hz.

In addition, respiratory time series (inductance plethysmography capturing both thoracic and abdominal excursions) were obtained at age 15. Gain for the respiratory leads was calibrated using a bag of known volume.

Subjects were first placed in the supine position and trained to breath in synchrony with a metronome at a frequency of 15 breaths per minute (0.25 Hz) to isolate the heart rate variability generated by respiratory change from that arising in response to postural change, which naturally occurs at around 0.08 to 0.1 Hz, or about six times per minute. After a practice period, data was collected for four minutes in the supine position. Subjects were then asked to stand quietly while continuing to breath at the same frequency. After a brief period of equilibration of less than one minute, another four minutes of data were collected in this position.

Processing of Physiologic Data

The inter-beat (R wave to R wave) intervals of the surface ECG were digitized to within one millisecond. The beat series were visually inspected for artefacts. These were manually corrected using accepted methods designed to maintain the integrity of the time series (Cheung, 1981). In some instances, one or another, or both, of the supine or standing segments were unusable, and were discarded.

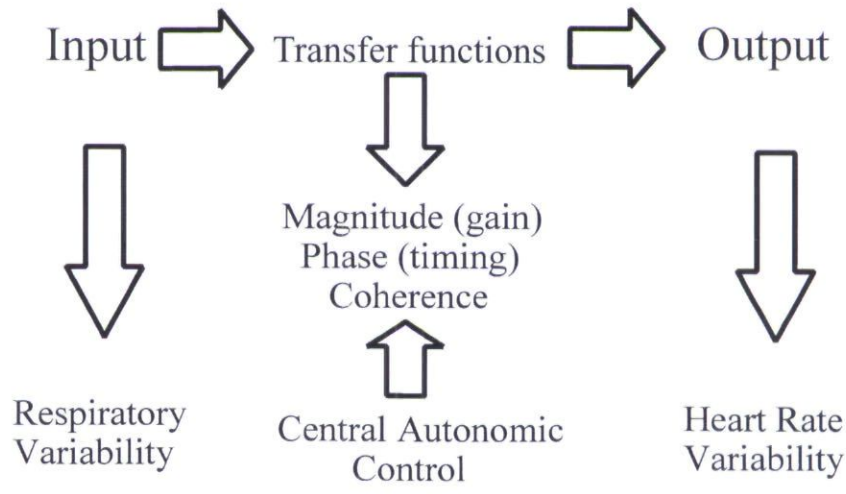

Figure 3. Schematic representation of the relationships between the respiratory source of heart rate variability, central autonomic control, and heart rate variability.

The respiratory signal was digitized at 5.0 Hz. Heart rate time series were reconstructed from the R–R interval series at 5.0 Hz, to match the respiratory time series (Berger, Akselrod, Gordon, & Cohen, 1986). From these two time series, corresponding two-minute segments were chosen from each of the supine and standing postures. Power spectra and transfer functions were generated from linearly detrended time series, using the Blackman-Tukey method (Berger et al., 1989; Saul et al., 1989, 1991). Power spectral and transfer function estimates were calculated for the respective mid- and high-frequency bands after four-point smoothing of the individual spectral point estimates, to reduce the error associated with these.

Two frequency bands were identified in each power spectrum: from 0.04 to 0.15 Hz, and from 0.20 to 0.30 Hz. These were labeled POST power (postural source), and RSA power (respiratory sinus arrythmia), respectively, to reflect their presumed origins. Estimates of respiratory transfer functions were taken at the prescribed breathing frequency of 0.25 Hz, or at the peak breathing frequency when this deviated from 0.25 Hz. When coherence of the transfer function was less than 0.5 (range 0.0 to 1.0), estimates of transfer magnitude and transfer phase were deemed statistically unreliable and were eliminated (Berger et al., 1989; Saul et al., 1989, 1991).

Statistical Analyses

To eliminate potential bias, subjects with a history of medical illness or treatment, including the use of psychotropic medications, were excluded from the analyses. Individual anxiety and antisocial behavior (or aggression) scores were transformed to *Z* scores at each assessment point. Individual composite scores for anxiety and antisocial behavior (or aggression) were generated by taking the respective means of the standard scores at all available assessment points.

Relationships between the composite anxiety and antisocial scores, and the cardiac indices at age 15, when both cardiac and respiratory data were available, were explored using stepwise regression models. Cardiac indices were predicted from the two behavior scores. Analyses were conducted separately for the supine and standing postures. Size (height and weight), maturation (pubertal status), and physical conditioning were included as predictors, if any one of them showed a significant bivariate relationship with the cardiac index of interest. The cardiac indices studied were mean heart rate, total heart rate variability, POST and RSA power, transfer magnitude, phase and coherence, and one derived variable, *balance*. This latter index represents the combined, reciprocal influences of the POST and RSA sources on heart rate variability (Equation 1) (Bootsma et al., 1994; Parati, Saul, DiRienzo, & Mancia, 1995; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

$$Balance = \frac{\text{POST power} - \text{RSA power}}{\text{POST power} + \text{RSA power}} \quad (1)$$

It varies from −1.0 for RSA dominance to 1.0 for POST dominance of heart rate variability.

Results

Subjects with heart rate data were 153 of the 175 boys. Missing cardiac data were the result of a computer malfunction. Further variations in the number of subjects for each analysis resulted because one or another segment of data was unusable, unreliable transfer function estimates were eliminated, medical illness or use of medications led to elimination of data, or behavioral data were incomplete. There were no differences in the mean individual anxiety or antisocial behavior scores for subjects with complete heart rate and behavioral data, and the remainder of subjects without heart rate data from the original study.

Before proceeding to analyses germane to the questions of our study, some characteristics of the behavioral and cardiac indices themselves are presented.

Relationships among Behavioral Scales

The alpha coefficients for the items of the antisocial and anxiety scales for subjects in the original study were as follows; antisocial scale: age 10, .87 ($N = 984$); age 11, .88 ($N = 914$); age 12, .89 ($N = 897$); age 13, .89 ($N =$

Table 1
Cardiac Indices, Units of Measure, Means, and Standard Deviations for Entire Sample

Cardiac index	Supine	Standing	Change
Mean heart rate (bpm)	69.87 (10.19)	96.74 (13.65)	27.12 (9.99)
POST/RSA balance[a]	−0.13 (0.46)	0.76 (0.20)	0.89 (0.44)
Total variability (bpm^2)	34.25 (27.82)	51.09 (29.94)	15.99 (36.30)
POST power (bpm^2)	8.08 (7.61)	24.51 (16.17)	15.94 (15.78)
RSA power (bpm^2)	11.57 (11.09)	3.24 (3.55)	−8.30 (9.95)
Transfer magnitude (bpm/liter)	35.74 (24.53)	14.97 (10.04)	−20.25 (22.49)
Transfer phase (degrees)	−3.91 (63.64)	−36.62 (63.73)	−32.78(62.53)
Transfer coherence[a]	0.80 (0.21)	0.63 (0.25)	−0.17 (0.25)

[a] Unitless indices.

854); age 14, .92 ($N = 817$); age 15, .92 ($N = 781$); anxiety scale: age 10, .54 ($N = 960$); age 11, .66 ($N = 906$); age 12, .67 ($N = 867$); age 14, .64 ($N = 826$). The alpha coefficients for the composite scales generated for the study sample were as follows; antisocial scale: .85 ($N = 181$); anxiety scale: .77 ($N = 185$).

Temporal stability for self-reported anxiety in the study sample varied from $r = .35$ ($N = 167$, $p < .0001$) for the 4-year interval from age 10 to age 14, to $r = .60$ ($N = 169$, $p < .0001$) for the 1-year interval from age 11 to age 12. Temporal stability for self-reported antisocial behavior varied from $r = .27$ ($N = 168$, $p < .0001$) for the 4-year interval from age 11 to age 15, to $r = .71$ ($N = 168$, $p < .0001$) for the 1-year interval from age 14 to age 15. The 5-year stability from age 10 to 15 was $r = .31$ ($N = 169$, $p < .0001$).

The composite self-reported anxiety score was not correlated with the composite self-reported antisocial behavior score. Neither maternal nor teacher reports of anxiety and aggression were independent of each other (teacher anxiety–aggression: $r = .37$, $N = 172$, $p < .0001$; maternal anxiety–aggression $r = .28$, $N = 170$, $p < .0003$).

Self-reported antisocial behavior was correlated with teacher ($r = .48$, $N = 171$, $p < .0001$) and maternal ($r = .31$, $N = 170$, $p < .0001$) ratings of aggression, whereas self-reported anxiety was not correlated with the ratings of either of these informants. Scores for self-reported anxiety and antisocial behavior were not related to height, weight, pubertal status, or physical conditioning.

Temporal Stability of Cardiac Indices

One-year temporal stability of cardiac indices between ages 14 and 15 were as follows: mean heart rate (supine: $r = .72$, $N = 110$, $p < .0001$; standing: $r = .37$, $N = 111$, $p < .0001$); total heart rate variability (supine: $r = .53$, $N = 109$, $p < .0001$; standing: $r = .25$, $N = 110$, $p < .01$); POST power (supine: $r = .37$, $N = 106$, $p < .0001$; standing: $r = .31$, $N = 109$, $p < .002$); RSA power (supine: $r = .37$, $N = 108$, $p < .0001$; standing: $r = .25$, $N = 109$, $p < .01$); and balance (supine: $r = .40$, $N = 106$, $p < .0001$; standing: $r = .44$, $N = 109$, $p < .0001$).

The one-year stability coefficient for standing heart rate found here is comparable to the one-year stability of the resting (seated) heart rate measure found for part of this sample when subjects were 9 and 10 years of age ($r = 0.41$, $N = 51$, $p < .01$) and 10 and 11 years of age ($r = 0.36$, $N = 51$, $p < .01$) (Kindlon et al., 1995). Murphy and colleagues (Murphy, Alpert, Walker, & Willey, 1991) found one-year temporal stability of resting heart rate in Caucasian boys between the ages of 9 and 10 to be similar to that of our sample at the same age ($r = .41$, $p < .01$, $N = 186$). The one-year stability of supine heart rate found here was considerably higher than the coefficients in both these studies, and was comparable to the one-year stability of antisocial behavior.

Comparable data on the stability of resting (seated) spectral indices in adults has been presented by Sloan and colleagues (Sloan, Shapiro, Bagiella, Gorman, & Bigger, 1995). The average age of subjects ($N = 20$) in this study was 46.1 years (SD 10.1). Repeat measures were taken at 3- and 9-month intervals from the initial assessment, and stability was calculated using the intraclass correlation coefficient. They found that heart rate (0.78), the natural logarithm (ln) of total power (0.70), ln low-frequency power (0.76), ln high-frequency power (0.84), and the low-frequency/high-frequency ratio (analogous to balance) (0.86), were all highly stable under the experimental circumstances in this older sample.

Effects of Postural Change on Cardiac Indices

Matched pair t-tests comparing the supine and standing values of corresponding cardiac indices showed that postural change exerted a significant effect on all of these. Indices that increased with postural change were heart rate ($t = 29.6$, $N = 119$, $p < .0001$), total heart rate variability ($t = 4.77$, $N = 117$, $p < .0001$), POST power ($t = 10.79$, $N = 114$, $p < .0001$), and POST/RSA balance ($t = 21.71$, $N = 114$, $p < .0001$). Indices that decreased with postural change were RSA power ($t = -8.98$, $N = 116$, $p < .0001$), and all three transfer function estimates: transfer magnitude ($t = -8.51$, $N = 85$, $p < .0001$), transfer phase ($t = -4.73$, $N = 85$, $p < .0001$), and coherence ($t = -7.61$, $N = 118$, $p < .0001$). All changes were in the expected direction. Table 1 summarizes the means, standard deviations, and units of measurement for the supine, standing, and change indices, for the entire sample.

Table 2
Parameter Estimates for Cardiac–Behavior Relationships, Including the Effects of Height, Weight, Pubertal Status, and Physical Conditioning Where Pertinent, for Entire Sample

Cardiac index	Predictor	Parameter estimate	Standard error	F	p
Supine heart rate	Height	−0.33	0.11	8.27	.005
	Anxiety	3.07	1.16	6.98	.010
	Antisocial	−2.21	1.15	3.68	.060
Standing heart rate	Anxiety	4.02	1.45	7.69	.007
	Antisocial	−3.95	1.46	7.33	.008
Supine balance	Antisocial	0.14	0.06	6.20	.020
	Phys. cond.	0.02	0.01	3.71	.060
Standing balance	Anxiety	0.06	0.02	7.95	.006
	Phys. cond.	0.01	0.005	6.31	.020
Supine total variability	Antisocial	−5.78	3.22	3.22	.080
	Height	−0.57	0.34	2.78	.100
	Weight	−0.29	0.19	2.26	.140
Standing POST power	Anxiety	3.71	1.81	4.17	.050
Supine RSA power	Antisocial	−4.06	1.29	9.84	.002
Standing phase	Anxiety	−17.63	8.48	4.32	.040

Relations of Behavioral Characteristics to Cardiac Indices

Although data from multiple informants was obtained, we chose to focus on the relationships between self-reported anxiety and antisocial behavior and the cardiac indices, for the following reasons. Correlations across informants were moderate for the antisocial/aggressive behaviors, but absent for anxiety. Self-reported anxiety and antisocial behaviors were independent of each other, whereas anxiety and aggression for both maternal and teacher reports were not. Anxiety is highly subjective, and in the absence of discovery or apprehension, parents and teachers would also be unaware of many adolescent antisocial behaviors. That adolescents are valid and reliable reporters of their own aggression, delinquency, and anxiety, has been demonstrated (Achenbach, Howell, McConaughy, & Stanger, 1995a, b; Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985).

Outcomes for the analyses did not differ whether raw power indices or the natural logarithm of power was used for the POST and RSA indices. Data for the raw power indices are reported. In this way data were not lost if zero values for these indices, or for balance, led to missing logarithmic values.

Anxiety

Increasing levels of anxiety were related to increasing heart rate in both the supine and the standing postures [*supine heart rate*: parameter estimate 3.07, $F(3,118) = 6.98$, $p < .01$; *standing heart rate*: parameter estimate 4.02, $F(2,121) = 7.69$, $p < .007$]. Increasing levels of anxiety were also related to increasing *POST/RSA balance* in the *standing* posture [parameter estimate 0.06, $F(2,115) = 7.95$, $p < .006$]. Consistent with this, increasing levels of *POST power* in the *standing* position were noted [parameter estimate 3.71, $F(1,120) = 4.17$, $p < .05$]. This relationship held even when POST power was normalized relative to units of total heart rate variability [$F(1,120) = 4.98$, $p < .03$], to account for individual variations in the latter across individuals (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Regarding the transfer of respiratory to heart rate variability, the *phase* or timing of the heart rate response to respiratory changes was increased in the *standing* posture (i.e. there was increased time lag in the transfer) in relation to increasing anxiety [parameter estimate −17.63, $F(1,90) = 4.32$, $p < .04$).

Antisocial Behavior

Increasing levels of antisocial behavior were related to diminishing heart rate in the standing posture, and to a trend for the same in the supine posture [*supine heart rate*: parameter estimate 2.21, $F(3,118) = 3.68$, $p < .06$; *standing heart rate*: parameter estimate 3.95, $F(2,121) = 7.33$, $p < .008$]. Increasing levels of antisocial behavior were also associated with increasing *POST/RSA balance* in the *supine* posture [parameter estimate 0.14, $F(2,111) = 6.20$, $p < .02$]. Consistent with this shift in balance, reductions in *supine RSA power* [parameter estimate −4.06, $F(1,118) = 9.84$, $p < .002$] were noted. This reduction in supine RSA power was also evident in a trend for reduced total heart rate variability in the supine posture [parameter estimate −5.78, $F(3,117) = 3.22$, $p < .08$]. Nonetheless, the relationship between RSA power and antisocial behavior remained even when RSA power was normalized relative to units of total heart rate variability [$F(3,117) = 6.58$, $p < .01$].

Summaries for the models relating behavior characteristics to the cardiac indices are presented in Table 2.

Table 3
Group Comparisons for Mean Heart Rate, Respiratory-driven Heart Rate Variability, and Respiratory to Heart Rate Transfer Function Estimates in the Supine Posture

Group	Mean heart rate	Total heart rate variability	RSA power	Transfer magnitude 0.20–0.30 Hz	Coherence 0.25 Hz
Controls	71.81	52.51	20.65	44.01	0.90
Anxious	73.14	32.86	13.83	40.23	0.78
Antisocial	62.05	21.04	4.18	24.50	0.70

Post Hoc Analyses

Because respiratory-driven heart rate variability was found to be reduced in the supine posture in relation to increasing levels of antisocial behavior, and because this was not related to decreasing levels of transfer magnitude at 0.25 Hz, a stepwise regression examining the relationship of antisocial behavior to supine respiratory power or drive over the RSA frequency band (0.20 to 0.30 Hz) was conducted. No relationship was found between the antisocial behavior score and respiratory power over this frequency range. Therefore, the reduced RSA power in relation to increasing antisocial behavior score was not due to diminished respiratory drive, the principle source of this component of the heart rate variability.

Subsequently, homogenous groups of antisocial only, anxious only, and neither antisocial nor anxious subjects were created, in a further attempt to elucidate the nature of the reduced RSA power associated with increasing antisocial behavior scores. To be included in either the antisocial ($N = 10$) or the anxious ($N = 14$) category, subjects had to have inclusionary scores above the 75th percentile on the one scale, and exclusionary scores below the mean on the other. Subjects who were neither anxious nor antisocial ($N = 44$) had scores below the mean on both scales. In order to generate a control group ($N = 8$) of similar size to the other two groups, scores on both scales below the 25th percentile were used.

Table 3 summarizes the group comparisons for mean heart rate, and the respiratory–heart rate relationships in the supine posture. Mean heart rate [$F(2,29) = 4.58, p < .02$], total heart rate variability [$F(2,29) = 3.97, p < .03$], RSA power [$F(2,29) = 8.35, p < .002$], mean transfer magnitude of respiratory to heart rate variability from 0.20 to 0.30 Hz [$F(2,29) = 1.73, p < .20$), and coherence between the respiratory and heart rate time series at the principle respiratory frequency [$F(2,29) = 2.05, p < .15$], were all lowest in the antisocial group. Here again, there were no group differences in the amount of respiratory power or drive over the RSA frequency band.

Of primary interest is the trend for transfer magnitude to be reduced in the RSA region of the power spectrum in antisocial individuals. A repeated measures analysis of mean transfer magnitude as a function of posture showed an even stronger trend for a Group by Posture interaction ($F = 2.66, p < .09$), due to the fact that antisocial subjects as a group started at lower levels of transfer magnitude, and therefore had the smallest change in this index in relation to postural change (Table 4).

The group comparisons helped elucidate the nature of the relationship between levels of antisocial behavior and supine RSA power. The central autonomic transfer of respiratory to heart rate variability appeared to be reduced in antisocial individuals. This impression is supported by the trend for there to be reduced coherence between the two time series in antisocial individuals as well.

Table 4
Group Comparisons for Mean Respiratory to Heart Rate Variability Transfer Magnitude in the Supine and Standing Postures

Group	Supine transfer magnitude (bpm/liter)	Standing transfer magnitude (bpm/liter)
Controls	44.01 ($N = 8$)	12.29 ($N = 8$)
Anxious	40.23 ($N = 14$)	11.76 ($N = 15$)
Antisocial	24.52 ($N = 10$)	13.66 ($N = 12$)

Repeated measures Group by Posture interaction, $F = 2.66$, $p < .09$.

Discussion

We studied the relationship of heart rate regulation to anxiety and to antisocial behaviors in a homogenous sample of adolescent boys. We took into account medical history, and the effects of body size, pubertal maturation, and physical conditioning when examining these relationships. We measured heart rate and respiration, and utilized two physiologic challenges, paced respiration and postural change, each designed to elicit specific sources of heart rate variability. Contributions to the heart rate variability from each source, and estimates of the balance between them, were examined as a function of the behavioral characteristics. The central autonomic processing of the transfer of respiratory variability to heart rate variability was also studied in relation to these behavioral characteristics.

Our findings concerning the relationships of anxiety and antisocial behavior to heart rate were consistent with those of other investigators. The relationships for each of these with heart rate occurred in the expected direction. Increasing levels of anxiety were related to increasing heart rate in both postures, whereas increasing levels of antisocial behavior were related to decreasing heart rate in both postures.

The relationship of anxiety to regulation of heart rate variability indicated that heightened sympathetic mediation of phasic influences due to postural change were associated with increasing levels of anxiety. The balance between postural and respiratory sources of heart rate variability was shifted towards the former in the standing posture, largely as a result of the increases in POST power associated with higher levels of anxiety. The relationship

between anxiety and the timing of the transfer of respiratory to heart rate variability in the standing posture was yet another indicator of the heightened sympathetic mediatory role, since phase (timing) is delayed by the increasing sympathetic control found in the standing posture.

The relationship of antisocial behavior to the regulation of heart rate variability was not expected, and did not suggest heightened phasic vagal mediation of respiratory influences on heart rate. Instead, increasing levels of these behaviors were associated with a reduction of respiratory-driven heart rate variability in the supine posture, which was not accounted for by reductions in respiratory drive to the heart rate variability. Consistent with this reduction in RSA power, the balance of postural and respiratory influences in the supine posture were shifted towards the lower-frequency contributions in relation to increasing scores for antisocial behavior. The physiologic underpinnings of this reduction in vagally mediated phasic influences on heart rate appeared to be related to the central autonomic processing and transfer of respiratory variability to heart rate variability. Both the gain and the coherence of the relationship between respiration and heart rate showed trends to be reduced in antisocial individuals when homogenous subgroups were compared.

There is a growing body of literature relating vagally mediated heart rate control to the integrity of processes such as attention, cognitive competence, reactivity, and expression of emotion (Fox & Porges, 1985; Linnemeyer & Porges, 1986; Richards, 1987; Porges, Doussard-Roosevelt, & Maiti, 1994; Porges, Doussard-Roosevelt, Portales, & Seuss, 1994; Seuss, Porges, & Plude, 1994; Stifter, Fox, & Porges, 1989). The measures of cardiac vagal influence have been utilized as indirect quantitative indices of the status of these other central nervous system processes. Our finding concerning the relationship of antisocial behavior to RSA extends information of this sort into adolescence, in relation to processes of behavioral regulation.

These findings suggest that volitional control over phasic respiratory influences on heart rate is related to antisocial behavior in such a way that disruption of an ordinarily robust physiologic phenomenon is evidenced. Our findings do not, however, address whether reflexive control of spontaneous breathing and the ensuing effects on heart rate variability would be similarly related to antisocial behaviors. Evidence exists, in fact, that volitional control of respiration in felines operates in part via different autonomic pathways from that for reflexive control of spontaneous breathing (Orem, 1987, 1989). Nonetheless, the relationship of volitionally controlled RSA to antisocial behaviors is consistent with impressions that antisocial youths demonstrate altered control of other volitional behaviors that involve the higher cortical functions (Moffit, 1990; Seguin et al., 1995).

Returning to the hypothesis that certain forms of pathologic behavioral disinhibition, such as undersocialized conduct disorder, may be associated with reduced noradrenergic function, we did not find any direct, conclusive evidence for this in our study. Mean heart rate was reduced as a function of increasing antisocial behavior, but as noted before, this does not automatically imply reduced sympathetic (noradrenergic) influences. If, however, the reduction of phasic vagal influences is any indication of what occurs with tonic cardiac vagal control in antisocial individuals, then corresponding sympathetic influences would have to be reduced even more in order for heart rate to decrease as a function of antisocial behavior, thereby indirectly supporting the theory that conduct disorders are associated with reduced noradrenergic function, which in the case of our study is derived from a central autonomic measure. In fact, the stronger relationship between decreasing heart rate and increasing antisocial behavior was noted in the standing posture, a circumstance where sympathetic effects are normally more prominent.

From a methodological perspective, it bears mentioning that although indices of RSA have been widely used to represent cardiac vagal regulatory influences, and by inference the status of other central nervous system processes, postural indices have not been similarly utilized to represent cardiac sympathetic regulatory influences. This stems in part from the difficulty in attributing the mediation of the postural indices to only one branch of the autonomic nervous system. Nonetheless, our findings would indicate that under controlled circumstances designed to elicit particular physiological responses, postural indices of cardiac sympathetic control can also serve as useful noninvasive windows.

More importantly, perhaps, our findings suggest that indices derived from transfer function estimates may prove to be especially helpful where inferences regarding central autonomic processes are concerned. This was so, both for vagal and for sympathetic modulation of heart rate variability. Transfer function estimates take into account variations in the sources of the heart rate variability, and therefore should reflect more directly the central autonomic mediation between the physiological processes of interest.

It is noteworthy that neither anxiety nor antisocial behavior were necessarily related to the heart rate variability indices in exactly the same fashion in both postures. This underscores the importance of monitoring heart rate indices in different physiological states, and the likelihood that regulatory mechanisms may be differentially related to behavior depending on the state of the individual. It also points to the utility of choosing particular physiological provocations to elicit certain relationships.

The mean heart rate change of 27 bpm due to postural change (Table 1) requires comment. Older criteria based on adult samples suggested that changes greater than 20 bpm were indicative of dehydration. Based on the work of Streeten (1987), more recent guidelines issued by the American Heart Association (AHA) (Izzo & Black, 1993) suggest liberalized standards ($>$ 27 bpm change, or standing heart rate $>$ 109) even in adults. There are no similarly established guidelines for pediatric samples, and it is expected that orthostatic changes will be more marked in younger subjects in relation to greater cardiovascular and vagal modulatory reactivity (Lipsitz, Mietus, Moody, & Goldberger, 1990). We have no reason to believe that our subjects were in general ill or dehydrated. Furthermore, since our data were gathered *within one minute* of assuming the erect posture, unlike

the guidelines of the AHA, which suggest that data be gathered at least *three minutes after* standing, we are confident that these heart rate changes are not the result of abnormal physiologic processes or aberrant data gathering.

In our protocol, we did not control for time of day, time lapse after meals, smoking history, or consumption of substances such as caffeine or nicotine immediately prior to the protocol; all of which could exert confounding influences on the physiological processes we studied. Another potential source of error, particularly in the estimation of transfer magnitude, was variation in calibration of the gain and balance of the two respiratory leads.

We did not quantify the transfer of variations in blood pressure to the heart rate variability as we did for respiration, because we did not obtain a continuous measure of mean arterial pressure. This was largely due to the prohibitive costs of obtaining a device that measures mean arterial pressure reliably and noninvasively. If possible, however, subsequent investigations of the relationships of anxiety and antisocial behavior to heart rate regulation should examine these blood pressure transfer functions, as well as those related to the effects on heart rate of spontaneous breathing and controlled breathing at multiple frequencies.

Our experimental protocol was limited to two physiological challenges, so as to examine the relationships of particular behavioral patterns to the most basic physiological processes, before proceeding to other forms of challenge. Future studies of the relationships of anxiety and antisocial behavior to heart rate regulation should also explore the effects of cognitive, motivational, and emotional challenges.

Our sample was homogenous for age, sex, race, ethnicity, and socioeconomic status, permitting on the one hand control of these factors, but at the same limiting the generalizability of the results of this study. The study of these phenomena in both sexes, and in samples of diverse racial, ethnic, and socioeconomic extraction is needed. Similar protocols should be used to study these processes at earlier and later developmental stages. Longitudinal data is being collected on our sample, and will address the effects of developmental changes in late adolescence on heart rate regulation, and its' relationship to anxiety and antisocial behavior.

In summary, anxiety was predictably related to heart rate, to the sympathetic mediation of phasic postural effects on heart rate variability, and to the timing relationship between respiratory sources of heart rate variability and the heart rate variability itself. Antisocial behavior was predictably related to heart rate, but in contrast was unexpectedly related to disruption of the vagal mediation of RSA, a normally robust physiological phenomenon. This disruption appeared to take place at the level of the central autonomic processing between respiration and heart rate. Thus, anxiety and antisocial behavior showed distinct relationships to heart rate and to the autonomically mediated components of heart rate variability from postural and respiratory sources. Spectral analytic techniques aided our understanding of these relationships, suggesting utility for future research in this area.

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Appendix 1

Self-reported Antisocial Behavior

Have you threatened to beat up someone in order to make them do something they didn't want to do?
Have you had a fist fight with someone else?
Have you taken part in gang fights?
Have you beaten up someone who didn't do anything to you?
Have you carried a weapon (chain, knife, gun, etc.)?
Have you used a weapon in a fight with someone else?
Have you thrown rocks, bottles or other objects at anyone?
Have you stolen from home?
Have you stolen from school?
Have you shoplifted?
Have you kept an object worth less than $10.00?
Have you stolen a bicycle?
Have you sold stolen goods?
Have you kept an object worth between $10.00 and $100.00?
Have you stolen objects worth more than $100.00?
Have you been involved with breaking and entering?
Have you entered somewhere without paying?
Have you trespassed?
Have you taken drugs or alcohol?
Have you gotten drunk?
Have you destroyed school materials?
Have you been involved in vandalism at school?
Have you destroyed objects at home?
Have you vandalized a car?
Have you set a fire?

Items were rated on a scale from 1 to 4: never, once or twice, often, very often; and pertain to the previous 12 months, except at age 10 (see text).

Appendix 2

Self-reported Anxiety

I worry too much about whether I am doing the right thing.
I prefer to stay with the same group of friends.
I am easily hurt by criticisms or reprimands.
It is better not to confide in anyone.
I never tell lies.
I worry about what others think of me.
I am uncomfortable asking others for favors.
My heart beats rapidly when someone asks me too many questions.
I am afraid of many things, though I don't always show it.
I feel badly when someone criticizes me.
I wonder if others like me or not.
I would like to be less timid.
I have frequent headaches.
I am afraid to speak in front of my class.
From time to time I have stomach aches.
Most of the time I feel uneasy.
When things go wrong I usually think it is my fault.
I have trouble making up my mind.
It doesn't matter what I do, since I usually wonder whether I have done it well enough or not.
I am nervous and jittery.
When I am in trouble, it is usually my fault.

This scale measures perceived emotional discomfort in interpersonal relationships; i.e. emotionally uncomfortable in social situations, awareness of nervous tension (insomnia, other indicators of anxiety), shyness and sensitivity, and intropunitive (blames self and feels excessive guilt) attitudes. Items were scored as True/False, and pertain to the previous 12 months except at age 10 (see text).

Appendix 3

Maternal and Teacher Reports for Anxiety and Aggression

Aggression
Bullies other children
Kicks, bites, hits other children
Fights with other children

Anxiety
Is fearful
Seems distressed
Seems worried

Items are scored on a 3-point scale, and pertain to the previous 12 months.