Disinhibitory Psychopathology in Male Adolescents: Discriminating Conduct Disorder From Attention-Deficit/Hyperactivity Disorder Through Concurrent Assessment of Multiple Autonomic States

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T. P. Beauchaine (2001) recently proposed a model of autonomic nervous system functioning that predicts divergent patterns of psychophysiological responding across disorders of disinhibition. This model was tested by comparing groups of male adolescents with attention-deficit/hyperactivity disorder (ADHD) and attention-deficit/hyperactivity disorder plus conduct disorder (CD/ADHD) with controls while performing a repetitive motor task in which rewards were administered and removed across trials. Participants then watched a videotaped peer conflict. Electrodermal responding (EDR), cardiac pre-ejection period (PEP), and respiratory sinus arrhythmia (RSA) were monitored. Compared with controls, the ADHD and CD/ADHD participants exhibited reduced EDR. The CD/ADHD group was differentiated from the ADHD and control groups on PEP and from the control group on RSA. Findings are discussed in terms of the motivational and regulatory systems indexed. Implications for understanding rates of comorbidity between CD and ADHD are considered.

Conduct disorder (CD) and attention-deficit/hyperactivity disorder (ADHD) are highly comorbid conditions. Between 30% and 50% of children with ADHD also meet criteria for CD (Biederman, Newcorn, & Sprich, 1991). The proportion of CD cases with comorbid ADHD is higher still, often approaching 70% in clinical samples (Klein et al., 1997; Stewart, Cummings, Singer, & deBlois, 1981). During the 1980s, this extensive overlap generated much discussion in the child psychopathology literature (e.g., Hinshaw, 1987; Lahey, Green, & Forehand, 1980; Rutter, 1983), with some authors suggesting that CD and ADHD represent alternative manifestations of one underlying condition. Although a general consensus has since emerged that the disorders are at least somewhat distinct (see Biederman et al., 1991), comorbidity remains a topic of considerable interest, as efforts toward identifying the mechanisms responsible for the observed symptom overlap continue.

Of particular interest is the comorbidity between CD and the hyperactive/impulsive subtype of ADHD. This is because accumulating evidence suggests that CD accompanied by hyperactivity represents a particularly virulent condition, characterized by a strong genetic loading, increased rates of aggression, and elevated risk for future antisocial behavior (Faraone, Biederman, Jetton, & Tsuang, 1997; Faraone, Biederman, Mennin, Russell, & Tsuang, 1998; Lynam, 1996). Indeed, male adolescents selected for concurrent conduct problems and hyperactivity/impulsivity more closely resemble psychopathic than nonpsychopathic adult offenders (Lynam, 1998). These adolescents are more antisocial, more disinhibited, and score higher on measures of psychopathy than do adolescents with either disorder alone.

Subtyping more refractory cases of CD can be traced to the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980), which distinguished between probands low in impulsivity and aggression and an acutely disinhibited subtype characterized by a lack of empathy, failure to form loyal bonds with peers, and persistent use of instrumental aggression (see Quay, 1986). Although this subtype, referred to as undersocialized aggressive CD, has been dropped from the current nomenclature, several features composing it map onto the childhood-onset type represented currently in the DSM-IV (Lahey & Loebel, 1994). Childhood-onset CD is diagnosed when probands present with at least one criterion CD behavior prior to age 10. Members of this group are more aggressive than those of the adolescent-onset type (Waldman & Lahey, 1994). Moreover, their CD symptoms are typically preceded by hyperactivity and impulsivity (Loebel, Green, Keenan, & Lahey, 1995; Loebel & Keenan, 1994), with behavioral disinhibition playing a central role in the aggression exhibited by group members (see Walker, Lahey, Hynd, & Frame, 1987).

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Behavioral disinhibition is also a defining feature of hyperactive/impulsive ADHD (American Psychiatric Association, 1994; see also Barkley, 1994, 1997; Schachar, Tannock, & Logan, 1993; Schachar, Tannock, Marriott, & Logan, 1995). However, although probands with this diagnosis share impulsivity with the under- socialized aggressive and childhood-onset CD groups, 50% to 70% do not meet criteria for any CD subtype (see Biederman et al., 1991). Many are therefore not aggressive. Rather, their impulsivity is reflected in marked impatience, interruption of others, and difficulty regulating excitement. Both aggressive CD and hyperactive/impulsive ADHD are thus characterized by behavioral disinhibition, yet only in the former case is this disinhibition manifested in aggression. A challenge facing CD and ADHD researchers is to generate a theory that accounts for the impulsivity exhibited across these disorders and the differing rates of aggression observed between them. Unfortunately, much of the extant literature in this area is atheoretical. One exception that holds promise, however, has been offered by Jeffrey Gray (1982a, 1982b, 1987a, 1987b) and elaborated on by Herbert Quay (1988, 1993, 1997).

Gray’s Motivational Theory and Disinhibition

Gray (1982a, 1982b, 1987a, 1987b) outlined three interdependent brain systems, each subserving a distinct class of survival-related behaviors. Two of these have been linked to disinhibition, including the behavioral activation system (BAS) and the behavioral inhibition system (BIS). The BAS subserves appetitive motivational functions, governing approach and active avoidance. This system is responsible for maximizing rewards and for minimizing punishments in situations in which behavioral responses, and thus expenditures of energy, are required. The emotional byproducts of BAS activation include pleasure during approach and relief during active avoidance. In contrast, the BIS subserves aversive motivational functions, controlling passive avoidance and extinction. This system inhibits appetitive responding when aversive consequences are anticipated. The emotional byproducts of BIS activation are fear and anxiety. Gray proposed that the BIS is mediated centrally by the septo-hippocampal system, which is innervated by the serotonergic projections of the raphe nucleus and the noradrenergic projections of the locus ceruleus. In contrast, the BAS is mediated by the dopaminergic pathway including the ventral tegmental area, the nucleus accumbens, and the ventral striatum.

With differing degrees of success, Gray’s theory has been used in efforts to explain the disinhibition observed across CD and ADHD and the divergent rates of aggression observed between the disorders. Toward the former end, several authors have emphasized Gray’s assertion that the BAS and BIS are actively opposed to one another, with net output affecting behavior (see Fowles, 1980, 1988; Quay, 1993; Rogeness, Javors, & Pliszka, 1992). Disinhibition purportedly results from an imbalance in BAS and BIS functioning favoring behavioral activation. Thus, impulsivity occurs because punishment cues do not elicit sufficient anxiety to inhibit appetitive responding when competing reward contingencies are present. Theoretically, such outcomes could result from excessive BAS activity, attenuated BIS activity, or both.

Although the literature addressing this issue is voluminous and cannot be comprehensively covered here (for reviews, see Beauchaine, 2001; Fowles, 1980, 1988; Milich, Hartung, Martin, & Haigler, 1994; Nigg, 2000; Quay, 1988, 1993, 1997), several findings are germane to the present investigation. First, reduced norepinephrine precursors have been linked to aggressive CD (Rogeness, Hernandez, Macedo, & Mitchell, 1982; Rogeness, Hernandez, Macedo, Suchakom, & Hoppe, 1986; Rogeness et al., 1988) and to ADHD symptoms (Rogeness et al., 1986). In addition, ADHD groups exhibit reduced urinary norepinephrine metabolites (Shekim, Dekirmenjian, Chapel, & Davis, 1982; Shekim et al., 1987; Yu-cun & Yu-feng, 1984), and aggressive CD groups exhibit reduced cerebrospinal fluid serotonin metabolites (Krusel et al., 1990) compared with controls. Assuming that Gray’s proposition of noradrenergic and serotoninergic BIS mediation is correct, these findings are consistent with chronic BIS dysregulation in both disorders.

Second, Fowles (1980, 1988) reviewed evidence suggesting that BIS functioning is indexed by electrodermal responding (EDR). Consistent with the neurotransmitter data, both aggressive and ADHD children exhibit reduced EDR compared with controls, a finding that is well replicated (Delameter & Lahey, 1983; Iaboni, Douglas, & Ditto, 1997; McMarnott et al., 1993; Raine & Venables, 1984; Schmidt, Solanto, & Bridger, 1985; Zahn & Kruesi, 1993). Thus, on the basis of neurotransmitter and psychophysiological studies, both diagnostic groups appear to suffer from a deficiency in inhibitory motivation.

Nevertheless, Quay (1988, 1993, 1997) argued for different motivational profiles across disorders, attributing hyperactive/impulsive ADHD to attenuated BIS activity and aggressive CD to reward dominance or excessive BAS activity. Evidence offered for the reward dominance hypothesis is derived from response perseveration tasks, as initially used by Newman, Patterson, and Kosson (1987) with adult psychopaths. During such tasks, respondents typically play a card game in which they are rewarded monetarily for turning over winning cards and punished monetarily for turning over losing cards. The probability of winning at the beginning of the game is high but gradually decreases to zero. Reward dominance is indexed by the extent to which participants play into the decreasing schedule of reinforcement, thereby earning less money. In terms of support for the excessive BAS activity hypothesis in CD, results from these studies are mixed at best. Although CD participants perform more poorly than controls (e.g., Daugherty & Quay, 1991; Matthey, van Goospen, de Vries, Cohen-Kettenis, & van Engeland, 1998; Shapiro, Quay, Hogan, & Schwartz, 1988), they do not perform more poorly than participants with ADHD (Daugherty & Quay, 1991; see also Milich et al., 1994). Moreover, because these tasks include both reward and punishment cues, and therefore activate both motivational systems, they

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1 In his early writings, Gray referred to the behavioral activation system as the reward system (e.g., Gray, 1970). However, this label was dropped in favor of the term BAS, coined by Fowles (1980) and adopted eventually by Gray (see Gray, 1987a). More recently, Gray (Gray & McNaughton, 1996) has suggested that the term behavioral approach system be used.
probably do not index pure BAS activity. Thus, on the basis of these data, claims of reward dominance in CD but not ADHD appear to be somewhat overstated.

We have argued elsewhere that even if such motivational differences are confirmed, they cannot account fully for the behavioral disparities between aggressive CD and hyperactive/impulsive ADHD (e.g., Beauchaine, 2001). Although the motivational approach may account for disinhibition, the aggression exhibited by CD probands suggests a concurrent emotion regulation deficiency that cannot be explained solely by BAS and BIS functioning. As noted above, both CD and ADHD groups are impulsive, but only in the former case is this manifested in aggression. An emerging body of literature suggests that this aggression may be rooted in poorly regulated fight/flight responding (e.g., Davidson, Putnam, & Larson, 2000). Thus, a more inclusive model of disinhibitory psychopathology containing both motivational and regulatory components may be required to differentiate between CD and ADHD. Although Gray specified a fight/flight (F/F) system, he did not invoke it to explain externalizing behaviors. However, differences in F/F responding may indeed discriminate CD from ADHD probands.

Briefly, Gray (1987a, 1987b) proposed that the F/F mediates defensive reactions under conditions of frustration, punishment, and pain. The central substrates of the system include the amygdala, the periaqueductal gray matter, and the ventromedial hypothalamus. Activity within the F/F is determined in part by the emotional significance attributed to a stimulus. Thus, stimuli appraised as threatening are likely to activate the system. Because Gray suggested that the F/F produces aggression in a defensive capacity only, it has not been central to theories of CD, in which violence is often presumed to be driven instrumentally. However, early-onset conduct problems are also associated with reactive and thus defensive forms of aggression (Dodge, Lochman, Harnish, Bates, & Pettit, 1997). Group differences in F/F responding may therefore reflect the behavioral divergence observed between ADHD and aggressive CD probands. Moreover, these group differences may be indexed by cardiac vagal tone.

Porger's Polyvagal Theory, Emotion Regulation, and Aggression

The term vagal tone refers to parasympathetic influence on cardiac activity, as indexed by respiratory sinus arrhythmia (RSA) or cyclic increases and decreases in heart rate during respiration (Hayano et al., 1991; Katona & Jih, 1975). Because vagal outflow is inhibitory, increases in vagal efference during exhalation decelerate heart rate and decreases in vagal efference during inhalation accelerate heart rate. The latter occurs because excitatory sympathetic influences operate relatively unopposed during periods of vagal withdrawal.

As outlined by Porgeres (1995), vagal efferent fibers to the heart originate in both the dorsal motor nucleus and the nucleus ambiguus of the medulla. These autonomic efferents terminate on the sinoatrial node, the primary pacemaker of the heart. The dorsal motor nucleus controls the "vegetative" vagus, which mediates reflexive cardiac activity, including the deceleration of heart rate associated with orienting. This vagal branch is phylogenetically older and rooted in the primary coping strategy of reptiles, which freeze when threatened.

In contrast, the "smart" vagus, which mediates RSA, originates in the nucleus ambiguus and appears to have evolved in conjunction with the increased regulatory demands of complex social interaction (Porgeres, 1995). Thus, this vagal branch and RSA are both distinctly mammalian. After orienting to a conspecific, mammals must either engage with that social stimulus or initiate F/F responding. Engagement requires sustained attention, which is characterized by vagally mediated heart rate deceleration (e.g., Suess, Porgeres, & Plude, 1994; Weber, van der Molen, & Molenaar, 1994). In comparison, fighting and fleeing are accompanied by rage and panic, respectively, which are facilitated by near-complete vagal withdrawal, large sympathetically mediated heart rate accelerations, and fully attenuated RSA (George et al., 1989; see also Porgeres, 1995, 1996). Vagal withdrawal during intense emotional experiences is therefore functional, facilitating bursts of metabolic output to cope with real or perceived environmental demands. Thus, postorienting cardiac control, including heart rate acceleration associated with F/F activity, and heart rate deceleration associated with sustained attention and social engagement, is mediated by the nucleus ambiguous via the "smart" vagus (Porgeres, 1995).

At the structural level, projections from the hypothalamus and the periaqueductal gray matter to the medulla (see Heimer, 1995) provide links between F/F responding and vagal regulation of cardiac activity. Because medullary nuclei, including the nucleus ambiguus, represent the final common pathway from the central nervous system (CNS) to the autonomic nervous system (ANS), these structural relations also implicate the vagal system in modulating F/F responding. By way of inference, chronically angry, aggressive, and panic-prone groups may be characterized by reduced cardiac vagal tone, a prediction that has been confirmed empirically in antisocial male adolescents (Mezzacappa et al., 1996, 1997; Pine et al., 1998), trait hostile young adults (Sloan et al., 1994), and panic disordered probands (Yeragani et al., 1991, 1993). Each of these groups may be predisposed to F/F responding because of chronically attenuated vagal tone, which may mark a reduced threshold for acute anger and panic episodes. In comparison, and consistent with the hypothesis that the vagal system evolved to facilitate social engagement by regulating emotions, heightened basal RSA is correlated positively with social engagement of children in the classroom (Fox & Field, 1989), with teacher reports of social competence (Eisenberg et al., 1995), with expressions of empathy toward others in distress (Fabes, Eisenberg, & Eisenbud, 1993), and with efforts to provide assistance to others in distress (Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994).

Integrating the Motivational and Regulational Approaches

We recently proposed a model of ANS functioning that emphasizes the importance of both motivational and regulational determinants of behavior (Beauchaine, 2001). According

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2 Interested readers are referred to Porgeres (1995) for a more complete exposition of polyvagal theory.
Figure 1. An integrated model of autonomic nervous system–behavior relations in which motivational functioning is mediated peripherally by the sympathetic nervous system, and regulatory functioning is mediated peripherally by the parasympathetic nervous system. The effects on target organs of increased activity in each system are indicated by solid lines and arrows. Note that behavioral and emotional predispositions cannot be predicted by activity in any single system, as each provides an independent contribution (dashed lines and arrows). Note also that the figure is highly schematic, with structures omitted that are not related directly to the systems under consideration.

to this model (see Figure 1), motivational predispositions, as reflected in the relative balance of the BAS and the BIS, are mediated peripherally by the sympathetic nervous system (SNS). In contrast, regulational functioning is mediated peripherally by the parasympathetic nervous system (PNS). Note that activity in both ANS branches marks behavioral and emotional predispositions. According to the presented model, both aggressive CD and hyperactive/impulsive ADHD groups are expected to exhibit a motivational deficiency that results in impulsivity (i.e., attenuated BIS functioning, excessive BAS functioning, or both). Thus, impulsivity is viewed as a consequence of a deficiency in motivational system functioning alone. In the case of pure ADHD, this impulsivity is buffered by adequate regulatory control of F/F responding, resulting in a normal or near-normal aggression threshold. In contrast, impulsivity in CD is expected to be met with inadequate F/F regulation, reflected in a reduced aggression threshold and attenuated RSA.

In other words, CD probands, with a lower threshold for F/F responding due to reduced “smart” vagal control, are less likely to engage with a social stimulus through deployment of sustained attention and more likely to exhibit impulsive aggression through deployment of F/F responding. This may help to

3 Although omitted from this discussion because of space limitations, it is worth noting that a parallel exists among the internalizing disorders in which anxiety is marked by excessive BIS activity, with panic disorder following if such activity is coupled with inadequate regulation of F/F responding, as indexed by RSA (Beachaine, 2001). Thus, dysregulated F/F responding is a nonspecific marker of both internalizing (panic) and externalizing (aggression) conditions. The form of such responding is hypothesized to be determined independently by inhibited or disinhibited motivational tendencies that are rooted in BAS/BIS profiles.
explain why CD but not ADHD probands are more likely than controls to interpret ambiguous peer behaviors as threatening and to endorse forceful means of dealing with such behaviors (e.g., Rabiner, Lenhart, & Lochman, 1990). Thus, although poorly regulated F/F responding does not determine aggression in any particular instance, it may result in CD probands being more likely to aggress.

A summary of predictions derived from the combined autonomic model regarding BAS, BIS, and emotion regulation system activity for both CD and hyperactive/impulsive ADHD is presented in Table 1. As outlined in the above literature review, several of the cell entries are supported empirically. Others, however, are largely speculative. The present investigation represents an effort to (a) test these more speculative predictions and (b) replicate the empirically supported relations through psychophysiological assessment of each biobehavioral system.

Before proceeding, however, it is necessary to elaborate on the conjecture that both the BAS and the BIS are mediated peripherally by the SNS. We have noted previously that Fowles (1980, 1988) has linked BIS functioning to EDR and that baseline EDR is reduced in cases of CD and ADHD (Delameter & Lahey, 1983; Iaboni et al., 1997; McBurnett et al., 1993; Raine & Venables, 1984; Schmidt et al., 1985; Zahn & Kruesi, 1993). In addition, in normative samples EDR increases under threat of punishment (Katkin, 1965; Kilpatrick, 1972) yet is largely unaffected by reward in well-controlled experiments (Iaboni et al., 1997; Tranel, 1983). These relations suggest that the BIS falls under SNS control because the eccrine sweat glands from which sudomotor activity originates are innervated by the SNS exclusively, with no parasympathetic input (see Fowles, 1986).

Evidence tying BAS functioning to the SNS 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 et al., 1990; Sherwood, Allen, Obrist, & Langer, 1986). Third, the reticular nuclei that control PNS influences on cardiac functioning are phylogenetically new developments, with maximum differentiation in the mammalian brainstem (Porges, 1995). 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-linked cardiac activity. Consistent with this hypothesis, larger increases in heart rate are observed when participants respond for monetary incentives than when no incentives are present (see Fowles, 1980, 1988; Iaboni et al., 1997). However, heart rate cannot be used as a pure index of SNS activity because it is affected by both ANS branches, which can operate with considerable independence (see Berntson, Cacioppo, Quigley, & Fabro, 1994). To date, SNS-specific measures of cardiac functioning have not been used in the disinhibition literature.

Note that in addition to suggesting that the SNS mediates both BAS and BIS functioning, the above discussion also implies differentiation within the SNS under appropriate stimulus conditions. Thus, BAS activity may be indexed by SNS-linked cardiac activity during reward, and BIS activity may be indexed by EDR during extinction of previously rewarded behaviors.

Aims of the Present Study

Following from the above discussion, the primary objective of the present investigation was to compare aggressive CD, hyperactive/impulsive ADHD, and control groups of adolescents on psychophysiological indices of BAS, BIS, and regulatory functioning. As summarized in Table 1, three general hypotheses were tested. First, we hypothesized that CD participants would exhibit elevated BAS activity compared with both ADHD participants and controls, as indexed by SNS-linked cardiac activity (see below). This hypothesis was considered somewhat speculative given the inconsistencies in the literature noted above. Second, we hypothesized that both the CD and ADHD groups would exhibit less BIS activity than controls, as indexed by EDR. Third, we hypothesized that CD participants would exhibit reduced vagal tone compared with both the ADHD and control groups, as indexed by RSA.

Each of these group differences was expected to be observed during baseline conditions. Although it has been suggested that BAS activity can be indexed specifically during reward, and that BIS activity can be indexed specifically during extinction (e.g., Fowles, 1980, 1988), baseline activity levels of many biological systems mark the efficiency of those systems in responding to stress. Resting heart rate, for example, differentiates between fit and nonfit people and marks efficiency of the cardiovascular system to respond to exercise.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Predicted Patterns of Functioning in Three Biobehavioral Systems for Disorders of Disinhibition</th>
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</thead>
<tbody>
<tr>
<td>Measure</td>
<td>Biobehavioral system</td>
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<tr>
<td>Function</td>
<td></td>
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<tr>
<td>Behavioral predisposition</td>
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<tr>
<td>Regulated impulsivity (ADHD)</td>
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<tr>
<td>Unregulated impulsivity (CD)</td>
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<tr>
<td>Normal?</td>
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<tr>
<td>Low</td>
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<tr>
<td>High?</td>
<td></td>
</tr>
<tr>
<td>Low</td>
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</table>

Note. Entries with question marks are those for which little or no empirical support exists. All other entries are supported empirically, as outlined in the preceding literature review. F/F = fight/flight; ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; High = atypically high activity; Low = atypically low activity.
Moreover, large increases in heart rate are observed during strenuous activity in both groups. Consistent with this reasoning, disinhibited children and adolescents exhibit reduced EDR compared with controls at baseline, during mild punishment, in response to orienting stimuli, and in response to mental stressors (Delamater & Lahey, 1983; Iaboni et al., 1997; McBurnett et al., 1993; Raine & Venables, 1984; Schmidt et al., 1985; Zahn & Kruesi, 1993). Thus, the utility of EDR as a marker of disinhibition does not appear to be confined to conditions of punishment or extinction.

An additional objective was to extend these assessments to stimu-
lus conditions of specific theoretical relevance to each biobehavioral system. Thus, groups were also compared on measures of reactivity while participating in several experimental tasks. These included (a) responding repetitively for monetary incentives, a reward condition intended to elicit BAS (SNS-linked cardiac) reactivity; (b) responding repetitively during incentive removal, an extinction condition in-
tended to elicit BIS (EDR) reactivity; and (c) viewing a provocative peer interaction, a social threat condition intended to elicit vagal (RSA) reactivity. Greater BAS reactivity, reflected by increases in SNS-linked cardiac measures, was predicted during reward by the CD group compared with ADHD and control participants. Following from previous research (Iaboni et al., 1997), reduced BIS reactivity, reflected in smaller EDR changes, was predicted during extinction by both CD and ADHD participants compared with controls. Finally, escalating vagal withdrawal, reflected in reduced RSA, was expected in all groups as the peer conflict progressed. Group differences in reactivity were therefore not predicted for this measure. However, the CD group was expected to maintain RSA deficits observed at baseline, reflecting larger functional reductions in vagal tone, and increased risk for F/F responding.

Method

Participants

The sample included 22 control, 17 ADHD, and 20 aggressive CD male adolescents between the ages of 12 and 17 years. This sample size was based on a set of power analyses conducted on published reports assessing group differences between controls and either CD or ADHD participants in EDR (Iaboni et al., 1997; McBurnett et al., 1993), RSA (Mezzacappa et al., 1996, 1997), and heart rate (see McBurnett & Lahey, 1994). No studies assessing group differences in PEP were available. All participants were recruited through advertisements placed in local newspapers and community publications covering approximately a 10-mile radius surrounding the State University of New York at Stony Brook. Three separate ads were used, targeting male adolescents who were “well adjusted,” “hyperactive,” or “experiencing behavior problems.” Each of the ads apprised parents that they and their son could earn up to $25 for a 1-hr visit to the university psychology department.

Parents who responded completed a 30-min phone interview with a trained research assistant who administered a computerized diagnostic instrument that included portions of the Adolescent Symptom Inventory (ASI; Gadow & Sprafkin, 1997) and the Child Behavior Checklist (CBCL; Achenbach, 1991). The ASI yields both dimensional scores and diagnostic cutoffs for many DSM-IV disorders. Symptoms are rated on a 4-point scale (0 = never, 1 = sometimes, 2 = often, 3 = very often), with ratings of 2 or higher considered positive for a given diagnostic criterion. Sensitivity and specificity of the scales used in this study are adequate to excellent (Gadow & Sprafkin, 1997). Nevertheless, the CBCL was also administered as a source of convergent diagnostic information. A total of 256 parents completed the ASI Conduct Disorder, ADHD, Oppositional Defiant Disorder (ODD), and Major Depressive Disorder (MDD) scales, as well as the CBCL Aggression, Hyperactivity, and Delinquent Behavior scales. To ensure that groups of aggressive CD and hyperactive/impulsive ADHD adolescents were recruited, several exclusionary criteria were used. Cases were rejected from the experimental groups if their child (a) did not meet criteria for CD or ADHD, (b) met criteria for CD without aggressive symptoms, (c) met criteria for ADHD without impulsivity, (d) met criteria for ADHD and ODD, or (e) met criteria for MDD. The final two groups were excluded because CD is often preceded by ODD (e.g., Biederman et al., 1996) and because depression appears to moderate the relationship between RSA and aggression (Beauchaine, Gartner, & Hagen, 2000). Those adolescents meeting DSM-IV diagnostic criteria for CD were also required to obtain T scores placing them at or above the 95th percentile on both the Delinquent Behavior and Aggressive Behavior scales of the CBCL, in reference to national norms. Those adolescents meeting DSM-IV criteria for ADHD were required to (a) score at or above the 95th percentile on the CBCL Attention Problems scale, (b) score below the 70th percentile on the CBCL Delinquency scale, and (c) endorse no CD diagnostic criteria. Potential control group parents received identical interviews and were excluded if their sons met DSM-IV criteria for any disorder or if they obtained T scores on any of the CBCL scales placing them above the 60th percentile. All instruments were scored by computer immediately, and invitations to participate were extended to parents whose sons met the inclusion criteria for one of the three groups. This invitation included a $40 monetary incentive, and parents were informed that their son could earn an additional $10 to $20, depending on his performance playing a video game during the lab visit. Because of the potential impact of medications on the psychophysiological criterion variables, parents were asked to discontinue administering stimulants to their children 48 hours prior to their visit. Children who were administered an antidepressant were rejected from the study.

Sample descriptive statistics are summarized in Table 2. Consistent with the above discussion, the aggressive CD participants, although not selected for ADHD symptoms, exhibited mean hyperactivity levels placing them above the 98th percentile on the CBCL. All also met diagnostic criteria for hyperactive/impulsive ADHD as assessed by the ASI.4 From this point forward, this group is therefore referred to as the CD/ADHD group.

Task Conditions

Phase 1. Two experimental phases were used. In Phase I, patterns of cardiac and electrodermal activity were assessed at baseline and during conditions of reward for and extinction of repetitive responding. After consent forms were signed, participants were seated in a soundproof

4 Potential participants who were taking antidepressants were rejected be-
cause both tricyclics and tetracyclines attenuate RSA (Jakobsen, Hauksson, & Vestergaard, 1984; Mezzacappa, Steingard, Kindlon, Saul, & Earls, 1998; Yeragani et al., 1992) and because selective serotonin reuptake inhibitors have been reported to both increase (Tucker et al., 1997) and decrease (Rissanan, Naukkarinen, Virkkonen, Rawlings, & Linnola, 1998) parasympathetic tone. Moreover, unlike most stimulants, these medications can require weeks rather than hours to clear. Thus, asking participants to discontinue taking antidepressants was untenable. This resulted in the rejection of 4 potential participants.

5 At the outset of the study, a pure CD group was also sought. However, among the 256 potential participants whose parents were interviewed, only 1 met criteria for CD without hyperactivity/impulsivity. This difficulty in iden-
tifying a pure CD group is consistent with other reports in the CD literature (e.g., Klein et al., 1997). Although the inclusion of such a group would confer great opportunity for disentangling the automatic substrates underlying ADHD and aggression, testing the hypotheses set forth in the present study does not require a CD-only group. This is because the autonomic deficiencies predicted in CD fall over and above those predicted in ADHD. Moreover, examining autonomic responding in CD/ADHD participants is of practical significance, given the previously reviewed recalcitrance of their condition and given that their symptom profile occurs at a substantially higher base rate than pure CD.
Table 2
Sample Descriptive Characteristics by Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>ADHD (n = 17)</th>
<th>CD/ADHD (n = 20)</th>
<th>Control (n = 22)</th>
<th>F(2, 56)</th>
<th>( \eta^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>13.1 (1.2)</td>
<td>14.0 (1.6)</td>
<td>13.2 (1.3)</td>
<td>2.70</td>
<td>.09</td>
</tr>
<tr>
<td>Income (thousands of dollars)</td>
<td>64.9 (42.4)</td>
<td>49.9 (53.6)</td>
<td>73.5 (55.6)</td>
<td>1.17</td>
<td>.04</td>
</tr>
<tr>
<td>ADHD</td>
<td></td>
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<tr>
<td>ASI Hyperactivity scale score</td>
<td>18.7 (3.9)</td>
<td>14.9 (6.8)</td>
<td>2.3 (2.3)</td>
<td>64.86*</td>
<td>.70</td>
</tr>
<tr>
<td>No. of symptoms endorsed</td>
<td>7.4 (1.3)</td>
<td>4.9 (3.1)</td>
<td>0.3 (0.6)</td>
<td>66.78*</td>
<td>.70</td>
</tr>
<tr>
<td>ASI Inattention scale score</td>
<td>19.8 (4.9)</td>
<td>19.9 (5.3)</td>
<td>4.7 (3.5)</td>
<td>76.19*</td>
<td>.73</td>
</tr>
<tr>
<td>No. of symptoms endorsed</td>
<td>7.3 (2.4)</td>
<td>7.0 (2.2)</td>
<td>0.4 (1.0)</td>
<td>86.98*</td>
<td>.76</td>
</tr>
<tr>
<td>CBCL Attention Problems</td>
<td>78.6 (7.8)</td>
<td>74.8 (9.2)</td>
<td>51.1 (1.7)</td>
<td>90.95*</td>
<td>.76</td>
</tr>
<tr>
<td>CD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI Conduct Disorder scale score</td>
<td>1.3 (1.4)</td>
<td>11.9 (3.7)</td>
<td>0.2 (0.4)</td>
<td>155.69*</td>
<td>.85</td>
</tr>
<tr>
<td>No. of symptoms endorsed</td>
<td>0.0 (0.0)</td>
<td>4.1 (1.4)</td>
<td>0.0 (0.0)</td>
<td>171.75*</td>
<td>.86</td>
</tr>
<tr>
<td>CBCL Delinquent Behavior</td>
<td>54.8 (3.9)</td>
<td>73.7 (6.5)</td>
<td>51.5 (2.0)</td>
<td>143.99*</td>
<td>.84</td>
</tr>
<tr>
<td>CBCL Aggressive Behavior</td>
<td>64.8 (7.6)</td>
<td>79.5 (8.8)</td>
<td>51.1 (1.3)</td>
<td>94.98*</td>
<td>.77</td>
</tr>
</tbody>
</table>

Note. Groups with alternative subscripts differ significantly at \( p < .01 \). ASI scale scores represent the sum of dimensionalized symptoms (0 = never, 3 = very often). ASI symptom counts include the number of scale items endorsed at a level of 2 or higher (Gadow & Sprklin, 1997). CBCL data are expressed in \( T \) scores. ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; ASI = Adolescent Symptom Inventory; CBCL = Child Behavior Checklist.

* \( p < .001 \).

chamber that was monitored with a microphone and a video camera. Psychophysiological measures were first obtained during a 5-min baseline period. Participants then performed a computerized repetitive response task similar to that used by Fowles (1988) and Iaboni et al. (1997). This task was chosen for two reasons. First, reward and extinction are separated among trials rather than combined within trials as in other tasks presented in the literature (e.g., Newman et al., 1987). This provides for isolation of BAS activity during reward and BIS activity during punishment. Second, previous research using this task has produced group differences in psychophysiological responding but not in response speed (Iaboni et al., 1997). Thus, observed differences in autonomic activity cannot be attributed to differences in metabolic demands.

During the task, large, single-digit odd numbers (i.e., 1, 3, 5, 7, or 9) were presented to participants on a computer screen in random order. Participants were required to eliminate each image by depressing the matching digit on a 10-key pad with their dominant hand. They were then required to depress the Enter key (included on the 10-key pad), triggering presentation of the next stimulus. The key pad was fastened to a platform between the participants and the monitor so that hand movement only was required to depress the key for each digit. The task was performed across seven 2-min blocks, each separated by a 2.5-min rest period. Block 1 served as a practice period in which participants became familiar with the procedure. In Blocks 2, 3, and 4 (R1–R3), signal tones and 3\( \frac{1}{2} \) rewards accompanied correct responses, which were tracked by computer. A running total of money earned was presented continuously in the upper right corner of the computer screen. Rewards continued during the initial 30 s of Block 5 (R4), which was followed by 90 s of extinction in which signal tones and monetary incentives were removed (E1). Extinction of reward, including omission of signal tones, was initiated part way through this block to assess reactivity in the electrodermal and cardiac measures. Rewards and signal tones resumed for all of Block 6 (R5), with extinction reinstated at the beginning of Block 7 (E2). For the last 30 s of Block 7 (R6), rewards were reinstated so the procedure did not end in extinction. Participants were informed at the outset that rewards would be given on some but not all trials and that they needed to continue responding to know when rewards were available. The pattern of rewards was identical to that used by Iaboni et al. (1997). Payments were administered after completion of the entire protocol, including Phase II, described below. Psychophysiological measures were collected during each block and during the last 30 s of each rest period, when participants were required to sit quietly.

Phase II. In Phase II, group differences in electrodermal and cardiac reactivity were assessed while participants viewed an escalating conflict among peers. After a 2.5-min rest period following completion of the reward task, participants watched a 2-min videotape of two peers engaging in an argument that began with a minor disagreement and ended with one adversary pushing the other. The argument progressed through four 30-s phases, including (a) two male adolescents sitting in a waiting room reading magazines; (b) one adolescent asking the other to relinquish his magazine, a request that was met with mild verbal resistance; (c) the antagonist repeatedly demanding the magazine in a threatening tone; and (d) the two adolescents standing face-to-face yelling at one another, with one pushing the other as the video segment ended in a freeze-frame. These videotapes were filmed with both 13-year-old and 16-year-old actors. This enabled the age of actors to be matched to within 1 year of the age of participants.

Psychophysiological Measures

Non-specific fluctuations in skin conductance (NSFs). Skin conductance (SC) was recorded continuously using a Grass Model 7D polygraph (West Warwick, RI), as were all other psychophysiological measures. The SC signal was monitored by a constant voltage coupler (Lykken & Venables, 1971) with two standard 8 cm\(^2\) Ag–AgCl electrodes, secured to the thoracic eminence of the participant’s nondominant hand by using adhesive masking collars and a 0.05 molar NaCl electrolyte solution suspended in Parke-Davis Unibase cream (see Fowles et al., 1981). Non-specific skin conductance responses (SCRs) were scored as fluctuations exceeding 0.05 \( \mu \text{S} \).

Pre-ejection period (PEP). To assess SNS influence on cardiac activity, both electrocardiographic (ECG) and impedance cardiographic (ICG) signals were obtained. Sympathetic (beta-adrenergic) activity was indexed by PEP (McCubbin, Richardson, Langer, Kizer, & Obrist, 1983; Sherwood et al., 1990), or the time elapsed between the ECG Q-wave and the dZ/dt B-wave (i.e., the QBI interval; see below). Shorter intervals indicate
greater SNS activity. The specificity of PEP as an index of beta-adrenergic activity has been established through pharmacologic blockade (Sherwood et al., 1986). The ECG and ICG signals were obtained by using a Minnesota Impedance Cardiograph, Model 304B (Minneapolis, MN). Two tetrapolar aluminum/mylar tape electrodes were placed around the upper neck and two on the abdomen according to established guidelines (Sherwood et al., 1990). A 4 mA, 100 kHz alternating current was passed through the outer band electrodes. Transthoracic impedance (ZT) and the first derivative of pulsatile changes in transthoracic impedance (dZT/dt) were recorded by the inner electrodes. The ECG and ICG signals were synchronized to the nearest millisecond and saved to disk in 30-s epochs. Scoring was conducted by using an interactive computer program (Kelsey & Guethlein, 1990). Digitized ECG and ICG signals were inspected on a beat-to-beat basis and ensemble-averaged with respect to the ECG R-wave (Kelsey & Guethlein, 1990; Kelsey et al., 1998). In this scoring procedure, operators first view a display of the ICG waveforms and edit artifactual cardiac cycles. These cycles are excluded from the ensemble-averaging algorithm, which automatically scores five waveforms, three of which were used in this protocol. These included (a) the peak positive deflection of the ECG Q-wave, which signifies the onset of ventricular depolarization; (b) the peak negative deflection of the ECG R-wave, which signifies the peak of ventricular depolarization; and (c) the dZT/dt B-wave, which signifies the onset of ventricular ejection into the aorta.

Respiratory sinus arrhythmia (RSA). Vagal influences on cardiac activity were assessed using spectral analysis (see Bernston et al., 1997; Mezzacappa, Kindlon, Earls, & Saul, 1994). This involves decomposing the ECG R-wave time series into component heart rate variability frequencies by using fast-Fourier transformations. The resulting components are expressed in terms of a spectral density function, or the amount of spectral power within a given frequency band. For the purposes of this study, spectral power was divided into low- to mid-frequency variability (<0.15 Hz) and high-frequency variability (>1.5 Hz). Parasympathetic influences on heart rate, including RSA, are observed in the high-frequency range (Akselrod et al., 1981, 1985; Berger, Saul, & Cohen, 1989; Pomeranz et al., 1985; Saul et al., 1991; Saul, Berger, Chen, & Cohen, 1989). High-frequency spectral densities were calculated in 30-s epochs, using a software package developed by Richard Sloan and colleagues at Columbia University. All reported RSA values were normalized through log transformations.

Data Analyses

Mean SCRs, PEPs, and spectral densities were calculated for the final 2 min of the initial 5-min baseline, for the final 30 s of the baselines punctuating each block, and for the reward, extinction, and video conditions. To assess group differences in baseline responding, repeated measures analyses of variance (ANOVAs) were conducted for each psychophysiological index across the seven rest periods. Reactivity was assessed by conducting additional repeated measures ANOVAs using change scores in responding from (a) baseline to reward trials, (b) baseline to extinction trials, and (c) baseline to video phases. Significant group effects were followed up with appropriate planned comparisons, all of which were computed using pooled error terms. Effect sizes ($\eta^2$) for all such comparisons are reported. Group × Condition interactions, which were calculated using Greenhouse-Geisser-corrected degrees of freedom to adjust for departures from sphericity, were all nonsignificant and are thus not reported. Trend analyses are reported only for RSA, as this was the only variable for which such analyses were required to test experimental hypotheses. Although habituation trends were observed for both EDR and PEP across reward trials, these effects are omitted, as they were not central to the experimental hypotheses.

Task Performance

Before assessing psychophysiological response patterns, potential group differences in task performance were examined. Although such effects were not predicted and have not been observed in previous studies using this paradigm (e.g., Faboni et al., 1997), this strategy was necessary because differences in response speed could elicit concomitant differences in metabolic demands, providing a confound in analyses of group effects. Task performance was examined by first calculating participants’ average number of correct and incorrect responses per minute during each trial. Means and standard deviations are reported in Table 3. Due to hardware failure, task data were missing for two control participants. Potential group differences were assessed by conducting two 3 (Group) × 8 (6 Reward, 2 Extinction) repeated measures ANOVAs, one for correct and the other for incorrect responses. Group main effects and Group × Trials interactions were all nonsignificant. Post hoc contrasts revealed no differences in the number of correct responses or errors during reward versus extinction. The average amount of money earned by participants was $14.59 ($SD = $2.40) for the ADHD group, $15.65 ($SD = $2.60) for the CD/ADHD group, and $15.80 ($SD = $2.85) for the control group (in addition to the guaranteed family payment of $40). A one-way ANOVA revealed no group differences in the amount of money earned, $F(2, 54) = 1.17, p = .32, \eta^2 = .04.

Electrodermal Responding

Two predictions were offered regarding EDR. The first was that both ADHD and CD/ADHD probands would exhibit fewer SCRs at baseline, reflecting reduced tonic BIS activity. This hypothesis was tested by conducting a 3 × 7 (Group × Baseline) repeated measures ANOVA, which yielded a significant group effect, $F(2, 56) = 4.94, p = .01, \eta^2 = .15$. As predicted, planned comparisons indicated that both the ADHD group, $F(1, 56) = 9.17, p < .01, \eta^2 = .14$, and the CD/ADHD group, $F(1, 56) = 4.55, p < .04, \eta^2 = .08$, exhibited fewer SCRs than did controls. No difference was observed between the ADHD and the CD/ADHD groups, $F(1, 56) = 0.59, p = .45, \eta^2 = .01$. Baseline EDR is presented in the top panel of Figure 2. The second prediction regarding EDR was that ADHD and CD/ADHD probands would exhibit attenuated SCR reactivity dur-

* Selection of appropriate epoch lengths is an important consideration when using spectral analysis. One rule of thumb in the psychophysiology literature calls for a recording epoch that is 10 times the lowest frequency of interest (see Bernston et al., 1997). Others, however, have suggested that an epoch length of 4 times the lowest frequency of interest is sufficient (Mezzacappa et al., 1994). Analyses of respiration data collected during the present experiment revealed that all participants exhibited respiratory cycles above 0.20 Hz at baseline and above 0.30 Hz during the reward task. This suggests minimum epoch lengths of between 20 s and 50 s for the assessment of RSA at baseline and between 14 s and 33 s during task responding, depending on which of the above criteria is used. Because some of our task conditions were 30 s in duration, and because we were interested specifically in vagal reactivity, we chose an epoch length that fell between the two recommended minima.
Table 3

<table>
<thead>
<tr>
<th>Condition</th>
<th>Duration (s)</th>
<th>Correct responses per minute</th>
<th>Incorrect responses per minute</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>ADHD</td>
<td>CD</td>
</tr>
<tr>
<td>Reward 1</td>
<td>120</td>
<td>49.0</td>
<td>9.3</td>
</tr>
<tr>
<td>Reward 2</td>
<td>120</td>
<td>53.3</td>
<td>8.4</td>
</tr>
<tr>
<td>Reward 3</td>
<td>120</td>
<td>55.8</td>
<td>9.2</td>
</tr>
<tr>
<td>Reward 4</td>
<td>30</td>
<td>60.0</td>
<td>12.0</td>
</tr>
<tr>
<td>Extinction 1</td>
<td>90</td>
<td>53.8</td>
<td>9.2</td>
</tr>
<tr>
<td>Reward 5</td>
<td>120</td>
<td>57.1</td>
<td>9.7</td>
</tr>
<tr>
<td>Extinction 2</td>
<td>90</td>
<td>55.6</td>
<td>11.8</td>
</tr>
<tr>
<td>Reward 6</td>
<td>30</td>
<td>58.5</td>
<td>11.1</td>
</tr>
</tbody>
</table>

Note. ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder.

ing extinction, reflecting BIS insensitivity to punishment. This hypothesis was addressed by conducting a $3 \times 2$ (Group $\times$ Extinction Trials) repeated measures ANOVA in which change scores from baseline were entered. Contrary to prediction, no group effect was found, $F(2, 56) = 0.16, p = .85, \eta^2 = .006$, indicating that the ADHD, CD/ADHD, and control participants were not differentially reactive to extinction. Electrodermal reactivity to extinction is presented in the bottom panel of Figure 2.

Pre-Ejection Period

We offered two predictions regarding PEP. The first was that the CD/ADHD group would exhibit shorter PEPs at baseline compared with the ADHD and control groups. To test this hypothesis, a $3 \times 7$ (Group $\times$ Baseline) repeated measures ANOVA was conducted. Due to equipment failure, PEP data were missing for 1 CD/ADHD and 1 control participant. Results indicated a significant group effect, $F(2, 54) = 3.07, p = .05, \eta^2 = .10$. However, planned comparisons revealed that the CD/ADHD group exhibited longer PEPs than both the ADHD, $F(1, 54) = 4.59, p < .04, \eta^2 = .08$, and the control groups, $F(1, 54) = 4.32, p = .04, \eta^2 = .07$. Thus, contrary to prediction, CD/ADHD participants exhibited less SNS-linked cardiac activity than their ADHD and control counterparts. Baseline PEP data are presented in the top panel of Figure 3.

We also predicted that CD/ADHD probands would exhibit greater PEP reactivity to reward than the other groups. This hypothesis was tested by conducting a $3 \times 6$ (Group $\times$ Reward Trials) repeated measures ANOVA in which change scores from baseline were entered. A significant group effect was uncovered, $F(1, 54) = 3.16, p < .05, \eta^2 = .11$. Planned comparisons revealed that the CD/ADHD group exhibited less PEP reactivity than the control group $F(1, 54) = 6.30, p < .02, \eta^2 = .10$. Thus, again contrary to prediction, CD/ADHD probands exhibited less SNS-linked cardiac reactivity than controls in response to reward. No difference was observed between the CD/ADHD and ADHD groups, $F(1, 54) = 1.64, p = .21, \eta^2 = .03$. Pre-ejection period reactivity is presented in the bottom panel of Figure 3.

Respiratory Sinus Arrhythmia

The first hypothesis regarding RSA was that the CD/ADHD group would exhibit less baseline high-frequency spectral power than both the ADHD and control groups. This hypothesis was tested by conducting a $3 \times 7$ (Group $\times$ Baseline) repeated mea-
ures ANOVA,\(^7\) which yielded a significant group effect, \(F(2, 54) = 3.21, p < .05, \eta^2 = .11\). As predicted, planned comparisons indicated that the CD/ADHD group exhibited less high-frequency spectral power than the control group, \(F(1, 54) = 6.38, p = .01, \eta^2 = .11\). However, differences between the ADHD and CD/ADHD groups, \(F(1, 54) = 0.29, p = .59, \eta^2 = .02\), and between the ADHD and control groups, \(F(1, 54) = 1.77, p = .19, \eta^2 = .03\), were not significant. Baseline RSA data are presented in the top panel of Figure 4.

We also predicted that (a) all groups would exhibit diminishing RSA as the videotaped conflict progressed, and (b) baseline group differences would be preserved. Part (a) of this hypothesis was addressed by conducting a \(3 \times 4\) (Group \(\times\) Video Phases) repeated measures ANOVA in which change scores from baseline were entered. Consistent with predictions, no group effect was uncovered. However, a significant phase effect indicated reduced spectral power across trials for the sample, \(F(3, 162) = 2.94, p < .04, \epsilon = 0.87\). This effect was confirmed with a single degree of freedom trend analysis, \(F(1, 54) = 4.76, p < .04\). Thus, increasing vagal withdrawal was observed as the videotaped conflict escalated. Spectral power reactivity data are presented in the bottom panel of Figure 4.

We assessed the prediction that baseline group differences would be preserved by conducting a \(3 \times 4\) (Group \(\times\) Video Phases) repeated measures ANOVA using log-transformed RSA scores. As with the baseline data, a significant group effect was uncovered, \(F(2, 54) = 3.35, p = .04, \eta^2 = .11\). Consistent with predictions, the CD/ADHD group exhibited less high-frequency spectral power than both the control group, \(F(1, 54) = 5.67, p = .02, \eta^2 = .10\), and the ADHD group, \(F(1, 54) = 4.31, p = .04, \eta^2 = .07\).

**Discussion**

Three broad hypotheses were set forth in the introduction of this article, each following from predicted group differences in BAS.

\(^7\) There is some controversy over the need for statistical control of respiratory rate when assessing vagal influences on cardiac function. Grossman, Karemaker, and Wieling (1991) argued that respiration contributes to RSA independently of vagal influences and that heart rate variability attributable to respiration should be removed from estimates of RSA and vagal tone. However, frequency domain methods such as spectral analysis appear to be less affected by respiratory parameters than are methods that assess the amplitude of RSA oscillations (Grossman, van Beek, & Wijnen, 1990). Nevertheless, respiration data were collected in the present investigation across all experimental phases, using a Grass Model 7P (West Warwick, RI) volumetric low pressure transducer connected to a Physio 5 (Richmond, VA) pneumographic chest bellows. Although no group differences in respiration rate were observed, a parallel set of spectral power analyses was conducted in which vagal influences were estimated as the residual of high-frequency spectral power when predicted by respiration rate in bivariate regressions. All group differences were preserved, so only the simpler set of analyses (i.e., without respiratory control) are reported.
BIS, or emotion regulation system functioning and each with associated subhypotheses specific to baseline and task responding. In the sections to follow, we review these hypotheses and discuss the relevant findings. We then consider implications for current theories of disinhibition and for the combined autonomic model.

The first general hypothesis was that ADHD and CD/ADHD participants would exhibit attenuated EDR compared with controls, reflecting diminished BIS activity. This hypothesis was supported by the finding of reduced NSFs in both disinhibited groups at baseline compared with controls. However, the prediction that the CD and CD/ADHD groups would exhibit attenuated EDR reactivity during extinction was not supported, as no group differences were observed.

The second general hypothesis was that CD/ADHD participants would exhibit greater SNS-linked cardiac activity than would ADHD and control participants, reflecting excess BAS activity. This hypothesis was not supported. At baseline, CD/ADHD participants exhibited lengthened PEPs compared with the other groups, indicating attenuated rather than enhanced SNS activity. Also contrary to prediction, CD/ADHD probands exhibited less PEP reactivity during reward, again indicating reduced SNS-linked cardiac activity compared with the ADHD group and controls.

The third general hypothesis was that CD/ADHD group would exhibit reduced RSA compared to both the ADHD and control groups, reflecting reduced regulatory control and a lower threshold for F/F responding. This hypothesis was supported by the finding of attenuated RSA in CD/ADHD probands compared with controls at baseline. Moreover, this difference was maintained during presentation of the videotaped conflict, when CD/ADHD participants exhibited less RSA than both the ADHD and the control groups. Finally, the hypothesis of RSA reductions across escalating phases of the video was supported for the sample, indicating increased vagal withdrawal as the conflict intensified.

Implications for Current Models of Disinhibition

The finding of attenuated baseline EDR in both ADHD and CD/ADHD probands replicates previous reports of an inverse relation between electrophysiological activity and impulsivity (e.g., Delmater & Lahey, 1983; Iaboni et al., 1997; McBumett et al., 1993; Raine & Venables, 1984; Schmidt et al., 1985; Zahn & Kruesi, 1993). This finding may therefore appear to contribute little to our understanding of disinhibitory psychopathology. However, researchers assessing EDR in disinhibited samples have generally not used clearly defined CD and ADHD diagnostic groups. Thus, extant reports of electrophysiological hyporeactivity in ADHD could be attributable to comorbid CD, an interpretation that follows from a sizable body of literature linking reduced EDR to impulsive aggression and psychopathy (e.g., Fowles & Furuseth, 1994; McBumett & Lahey, 1994; Raine, 1996b; Scarpa & Raine, 1997). The present study resolves this ambiguity. Attenuated EDR characterized ADHD participants without CD as well as those with CD and appears to be a nonspecific marker of disinhibition. Given our previous argument that baseline differences in biological systems often mark the ability of those systems to respond to stress, we interpret this finding as consistent with the underactive BIS hypothesis in both disorders. However, group differences were not observed in EDR reactivity during extinction, despite Fowles’s (1980, 1988) contention that EDR reactivity to punishment reflects individual differences in BIS responding. Note that Fowles based this conjecture on within-subjects differences in EDR among non-psychiatric participants during extinction versus reward trials (see also Tranel, 1983). Moreover, no studies have addressed EDR reactivity during extinction in aggressive CD participants, rendering Fowles’s hypothesis largely untested in between-groups paradigms using psychopathological samples. In the lone study in which EDR was assessed during extinction, Iaboni et al. (1997) reported reduced reactivity in ADHD participants compared with controls, a finding that was not replicated in the present experiment. Because the task instructions and reward schedule used here were nearly identical to those described by Iaboni et al., it is unclear why similar group differences in EDR reactivity were not observed. One possibility is that participants did not experience the extinction trials as frustrating because they were informed that rewards would not always be provided. However, significant samplewide increases in EDR were observed during extinction compared with baseline, arguing against this interpretation. To clarify these discrepancies, further research assessing EDR during extinction in disinhibited probands is necessary.

The findings of reduced SNS-linked cardiac activity and reactivity in CD/ADHD probands also deserve elaboration, given that both ran counter to prediction. There are at least two possible explanations for these findings. One is that PEP is not a valid index of BAS activity and that the observed differences reflect an alternative underlying mechanism. This possibility cannot be ruled out with certainty. Perhaps equally plausible, however, is the possibility that PEP is a valid index of BAS activity and that the observed pattern of results reflects underactive BAS functioning in the CD/ADHD group. Although this post hoc interpretation is speculative, two sources of evidence may support it. First, significant PEP shortening was observed during reward trials in the control group, indicating increased SNS-linked cardiac activity. This finding would be expected if PEP marks normative shifts in BAS functioning during appetitive responding. Second, there is a sizable body of literature that can be construed as inconsistent with Quay’s (1988, 1993, 1997) proposition of increased BAS activity in aggressive CD/ADHD. Specifically, several authors have suggested that aggression represents a form of sensation seeking resulting from chronic underarousal (Eysenck, 1964; Eysenck & Gudjonsson, 1989; Quay, 1965; Zuckerman & Comro, 1983), as reflected in low resting heart rate (Raine, 1993, 1996a, 1996b; Raine, Venables, & Mednick, 1997). One interpretation of this literature is that reduced heart rate reflects insensitivity to reward and, by implication, an underactive BAS. Aggressive probands therefore engage in instrumental aggression to attain satisfactory reward states. Sensation-seeking formulations of disinhibition have a long history in the personality and aggression literatures. Thus, SNS influences on cardiac functioning may indeed index BAS activity, with the observed results supporting the stimulation-seeking hypothesis. By this reasoning, the finding of less PEP reactivity in CD/ADHD participants while responding for incentives is suggestive of BAS insensitivity to reward. This speculative account might be tested more directly by using larger incentives in future research to determine if PEP reactivity can be elicited during reward trials in CD/ADHD participants.
The reductions in spectral power observed across groups as the videotaped conflict escalated are consistent with the hypothesized evolutionarily functional relation between vagal withdrawal and F/F responding (George et al., 1989; see Porges, 1995, 1996). Thus, as the vignettes became increasingly threatening, we observed progressively greater vagal withdrawal. Recall the hypothesis that baseline group differences in vagal tone mark the capacity of the emotion regulation system to modulate F/F responding. If this hypothesis is correct, CD/ADHD participants, with reduced baseline vagal tone and a lower threshold for F/F responding, may indeed be at risk for aggression when task demands produce normative shifts in RSA (e.g., when attending or socializing).

Implications for the Integrated Model

The overarching thesis of this article was that both motivational and regulatory aspects of ANS functioning are required to explain the behavioral similarities and differences across and between subgroups of disinhibited probands. We predicted deficiencies in BIS functioning to account for the overlap in symptomatology in hyperactive/impulsive ADHD and aggressive CD/ADHD groups. We predicted concurrent deficiencies in BAS and vagal functioning in the CD/ADHD participants only, reflecting the motivational and regulatory substrates of their aggressive symptoms.

Findings of reduced EDR in both the CD/ADHD and the ADHD groups support the underactive BIS hypothesis in both disorders. Findings of lengthened PEP in the CD/ADHD group suggest attenuated BAS activity and may refute Quay’s (1988, 1993, 1997) claim of heightened BAS functioning in aggressive CD. Although speculative, these findings appear to be consistent with the sensation-seeking formulation of aggression and represent the first effort to discriminate between disinhibited subgroups based on SNS-linked cardiac activity. Previous reports have relied on heart rate to estimate SNS activity, an approach that is inadvisable because heart rate is influenced by both autonomic nervous system branches (see Berntson et al., 1994).

Findings of attenuated PEP activity in the CD/ADHD group may also refute the contention that the BAS and BIS are actively opposed to one another, with net output affecting behavior. Such an account implies that CD/ADHD participants, with reduced BAS and BIS activity, should not be impulsive. In contrast, the sensation-seeking hypothesis is not characterized by the active opposition assumption and may better account for the observed pattern of results. Of note, attenuated BIS functioning may nevertheless exacerbate the propensity for aggression in CD/ADHD probands, as sensation seeking is met with a disinclination to flight, reflected in reduced EDR. It bears repeating that these conjectures should be interpreted with caution, given the speculative nature of the sensation-seeking model.

The reductions in RSA observed in CD/ADHD participants are consistent with the proposed model and replicate recent reports of attenuated vagal tone in aggressive samples (Mezzacappa et al., 1996, 1997; Pine et al., 1998). This replication underscores the importance of considering emotion regulation deficiencies in accounting for aggressive behavior. However, reduced vagal influences alone cannot account for aggression, as similar reductions are observed in anxiety and panic disorders (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996; Yeragani et al., 1993). Thus, reduced RSA appears to be a nonspecific marker of dysregulated emotion and cannot be used in isolation to characterize aggressive probands. Motivational functioning must also be considered. As demonstrated here, aggressive participants also exhibit reduced BAS and BIS functioning. In contrast, anxiety and panic are marked by excessive BIS activity (see Beauchaine, 2001). Taken together, these findings suggest that the proposed model may be useful not only in distinguishing among disorders of disinhibition but also in differentiating between internalizing and externalizing disorders.

These findings also provide potential clarification regarding the high and asymmetric rates of comorbidity between aggressive CD and hyperactive/impulsive ADHD, as outlined at the outset of this article. Because ADHD probands are impulsive due to deficiencies in BIS functioning, typical degrees of vagal reactivity may place them at risk for emotional lability, reflected at times in aggressive behavior. Thus, CD symptoms might be expected to occur with some frequency in ADHD groups. Prevalence figures from both epidemiological and clinical samples support this supposition, with 30% to 50% of ADHD cases exhibiting comorbid CD (Biederman et al., 1991). Given the findings presented here, rates of comorbid ADHD in aggressive CD should be higher because the only dysregulated autonomic system observed in ADHD (i.e., the BIS) is also dysregulated in aggressive CD. Thus, aggressive CD probands should exhibit ADHD symptoms almost without exception, a prediction that is borne out empirically (see Klein et al., 1997).

This study does have some limitations. First, because the use of impedance cardiography with ADHD and CD/ADHD participants is new, future replications of these findings are imperative, particularly given the modest group sizes used. Moreover, because the sample was all male, generalizations cannot be made to female participants. It is known that normative samples of female subjects are characterized by heightened RSA compared with their male counterparts (Fabes et al., 1994; Lehofer et al., 1997; Stamps & Porges, 1975). Furthermore, RSA–behavior relations in female subjects may differ from those observed in male subjects (Eisenberg et al., 1995; Fabes et al., 1993), and relations between behavior and both EDR and PEP are largely unexplored in female samples. These issues should therefore also be pursued in future research.

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