Reduced Heart Rate Variability in Chronic Alcohol Abuse: Relationship with Negative Mood, Chronic Thought Suppression, and Compulsive Drinking

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Background: *Previous research suggests that heart rate variability (HRV) may be an important factor in various maladaptive psychological conditions.*

Methods: *This study was conducted to investigate vagal tone assessed as tonic vagally mediated heart rate variability in alcoholic (*n = *49) and control subjects (*n = *45).*

Results: *Alcoholic subjects had faster heart rate and lower preimaginary exposure levels of HRV compared with the control group. An increase in HRV was observed in the alcoholic group when subjects were exposed to an imaginary alcohol script. Tonic HRV was found to be related inversely to negative mood and chronic thought suppression and positively to positive mood. Furthermore, the compulsive subscale of the Obsessive Compulsive Drinking Scale (OCDS) was inversely related to HRV during the imaginary alcohol exposure.*

Conclusions: *It is concluded that the findings are in agreement with the neurovisceral integration model of affective regulation, which claims that dysfunctional psychologic states are rooted in an impaired inhibitory mechanism that is associated with low HRV.* Biol Psychiatry 2003;54:1427–1436 *© 2003 Society of Biological Psychiatry*

Key Words: Cue reactivity, thought suppression, craving, Obsessive Compulsive Drinking Scale, White Bear Thought Suppression Inventory, heart rate variability

Introduction

The recovering alcoholic must face the difficulty of having his or her ambition to remain abstinent challenged in various situations in which memories about the pleasurable effects of alcohol are activated and the striving for abstinence no longer seems meaningful (Anton 1999; Marlatt and Gordon 1985). The odds for successful coping with such temptations are related to numerous factors, such as one's subjective affective state and the ability to shift one's focus from the automatic impulse to drink toward a cognitive reconstruction of the situation (Palfai et al 1997b; Tiffany 1990). Despite the importance of attentional flexibility in effectively modulating such "high-risk" situations, research on the topic is scarce.

Cognitive neuroscience is a rapidly growing discipline that has as yet only been given a limited degree of the attention it deserves by mainstream addiction researchers. For example, the vast amount of research of self-reported craving has had limited success in explaining relapse to heavy drinking (Litt et al 2000; Tiffany and Carter 1998), although autonomic activity has sometimes proved useful in predicting drug use (Niaura et al 1989). Recently, however, an integrated physiologic model for understanding cognitive, emotional, and behavioral regulation has been proposed (Porges 1998; Thayer and Lane 2000). Thayer and Lane (2000) suggested that the interplay between positive (excitatory) and negative (inhibitory) feedback circuits in the nervous system (NS) allows for flexible and adaptive behavior across a wide range of situations. The uniqueness of this model lies with its emphasis on the importance of inhibitory processes in effective modulation of affective experience. In short, these researchers propose that the defects in neurovisceral regulation of affective experience seen in various psychiatric conditions (e.g., anxiety disorders) may be better explained by faulty inhibitory function in the NS than by unitary arousal models.

Tonic heart rate variability (HRV) may be a physiologic indicator of such inhibitory processes (Friedman and Thayer 1998a; Porges 1995). Heart rate variability refers to the complex beat-to-beat variation in heart rate produced by the interplay of sympathetic and parasympathetic (vagal) neural activity at the sinus node of the heart. Importantly, heart rate (HR) is under tonic inhibitory

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control via the vagus nerve (Levy 1990). These neural connections to the heart are linked to brain structures involved in goal-directed behavior and adaptability (Thayer and Lane 2000). Compelling evidence now exists to show that high levels of HRV are related to cognitive flexibility (Johnsen et al 2003), modulation of affect and emotion (see Bazhenova 1995, cited in Porges 1995), and increased impulse control (Allen et al 2000; Porges et al 1996).

The hypothesis that reduced HRV is related to defective affective and emotional regulation has been supported in recent research in which reduced HRV was present in clinical disorders such as generalized anxiety disorder (Thayer et al 1996), panic disorder (Friedman and Thayer 1998b), posttraumatic stress disorder (Cohen et al 1997), and depression (Thayer et al 1998). The role of such reduced HRV in alcohol abuse or addiction, however, has not been studied thoroughly. This state of knowledge is disappointing given the obvious conceptual relevance of inhibitory functions and impulse control in a disorder that has long been characterized by "loss of control" of voluntary behavior related to the relapse phenomenon (Jellinek 1960; Keller 1972; Lyvers 2000).

Indeed, several scientific arguments suggest that impaired inhibitory function may play a role in chronic alcohol abuse. First, alcoholics have repeatedly been shown to have problems shifting attention and directing their attention away from task-irrelevant information (Johnsen et al 1994; Setter et al 1994; Stormark et al 2000). Second, frontal areas of the brain are most affected by the acute and chronic effects of alcohol, and these structures are of crucial importance in inhibitory functioning and self-control (Lyvers 2000). Third, acute effects of alcohol ingestion result in reductions in HRV, implying that chronic alcohol ingestion may result in a long-lasting impairment of the vagal modulation of HR (Reed et al 1999; Weise et al 1986). Fourth, severely dependent alcoholics show a sustained phasic HR acceleration when processing alcohol information, indicating defective vagal modulation of cardiac function (Stormark et al 1998). Tonic HRV has similarly been found to be a useful measure of physiologic activity in challenging situations (Thayer and Lane 2000). Appropriate modulation of HRV (increases, decreases, or no change) depends on the type of challenge and the characteristics of individuals as they interact with specific contextual manipulation (Friedman and Thayer 1998a; Hughes and Stoney 2000; Porges et al 1996; Thayer et al 1996). For example, during attention-demanding tasks, healthy individuals show appropriate reductions in HRV (Porges 1995). In general, high tonic levels of HRV allow for the flexible deployment of organism resources to meet environmental challenges. With respect to attention, it is suggested that high levels of HRV reflect flexible attentional focus, whereas low HRV is related to "locked in attention" (Porges et al 1996). Moreover, increased tonic vagal activity is related to adaptive development and lack of behavioral and emotional problems (Hughes and Stoney 2000; Porges et al 1996).

If reduced vagal activity is associated with maladaptive psychiatric conditions, one could speculate that increased activity could be an indication of therapeutic effectiveness and recovery to healthier functioning. Several studies indeed suggest that increases in vagal activity as a result of treatment are a sensitive indicator of recovery from depression (Balogh et al 1993) and anxiety (Middelton and Ashby 1995). Furthermore, it has been demonstrated that increases in vagal activity during challenging tasks discriminates between individuals who have experienced traumatic events and managed to recover from them and those who still suffer from chronic symptoms of posttraumatic stress (Sahr et al 2001). Such increases in vagal activity during challenging tasks are particularly interesting because studies on alcohol abusers have found increases in HRV after exposure to alcohol-related cues (Jansma et al 2000; Rajan et al 1998). One could speculate that such enhanced vagal activity could be a sign of compensatory coping aimed at taming automatic drinking-related processes (Larimer et al 1999). Such an interpretation is in agreement with cognitive theories predicting that alcoholics and other drug users do not simply respond passively to exposure to drug-related cues, but, on the contrary, in such situations conscious processes are invoked, inhibiting execution of drug-related cognition (Tiffany 1990, 1995). If this explanation is correct, alcoholics who have more effective coping resources should show stronger increases in vagal activity during such challenging exposure than alcoholics who express greater difficulty in resisting drinking-related impulses. Unfortunately, however, such individual differences in impulse control were not reported in these studies, and thus such explanations remain highly speculative.

Nevertheless, studies suggest that individual differences are important in adaptive vagal functioning in processing of alcohol-related information (Stormark et al 1998), and treated individuals may sometimes manage to inhibit processing of drug-related information (Johnsen et al 1997; Stormark et al 1997). Thus, more research is needed to examine increases in HRV activity as a sign of activation of nonautomatic higher cognitive processes in alcoholics coping with "high-risk" situations.

Although general differences in HRV between alcoholics and nonalcoholics are interesting indicators of defective inhibitory functioning, it is necessary to assess the psychologic factors that are theoretically related to the neurovisceral integration model (Thayer and Lane 2000).

Table 1. Characteristics within the Alcoholic Group

Type of Measurement	Average[a]	No. Who Answered
Days Since Last Drink	81.5 (272.2)	46
Days Since Last Compulsive Drinking	79.6 (269)	46
Number of Standard Alcohol Units on the Last Drinking Day	29.56 (19.23)	47
Length of Last Drinking Period in Days	25 (40.47)	45
Craving Ratings	12.48 (7.5)	49
Obsessive Compulsive Drinking Scale, Total Score	26.73 (11.74)	46
Severity of Dependency Scale	9.25 (3.03)	47

[a]Values in parentheses are standard deviation.

An obvious candidate for such assessment is the measurement of affective tone to investigate whether dysphoric states are related to reduced vagal influences on heart rate and positive affective states are related to high vagal tone (Porges 1995). Furthermore, specific measures assessing the degree of impulse control related to drinking are needed to test whether such a reduced level of inhibition when processing alcohol-related information is related to low HRV (Anton 1999; Anton et al 1996). Moreover, a measure of rigid thought-control strategies and lack of cognitive control should be an important indicator of defective inhibitory function and "positive feedback loops" reflected as low HRV (Wegner and Zanakos 1994). Linking these measures to the physiologic index of HRV makes a stronger case for attributing reduced vagal tone (HRV) to a defective regulatory mechanism resulting in unpleasant affective states and maladaptive coping with psychologic stressors.

We therefore expected that alcoholics would have lower HRV than the control group and that HRV in alcoholics would increase after exposure to an alcohol-related imaginary script reflecting compensatory coping. Thus, we also expected that HRV in alcoholics would be negatively related to obsessive thoughts of alcohol and compulsive drinking. We expected that HRV would also be inversely related to negative affective states and rigid cognitive coping measures such as chronic thought suppression.

Methods and Materials

Participants

Forty-nine alcoholics (12 women) in treatment for alcohol abuse and 45 control subjects (14 women) participated in the experiment. The alcoholics were slightly older (M = 45.4) than the control subjects (M = 42), but this difference was not statistically significant. The alcoholic subjects were recruited from three inpatient treatment clinics at the Bergen Clinics Foundation and the Askøy Treatment Center as part of the Blue Cross substance abuse program. They had to be sober on the day of the testing and could not be experiencing acute withdrawal symptoms, psychoses, serious somatic disorders, or other complications. Further information related to the alcoholic subjects and measurements related to alcohol abuse are presented in Table 1. The use of other addictive substances including nicotine was not regarded as exclusion criteria, but alcohol abuse had to be the participant's primary reason for admission to the treatment program. Eighteen persons within the alcoholic group admitted some experience with substances other than alcohol and tobacco, but only two participants within the alcoholic group were identified as nonsmokers. Approximately half of the alcoholics ($n = 23$) took psychotropic medication, but only five participants were taking medication that could in some exceptional cases have cardiovascular side effects.

The control participants were recruited primarily from a telephone survey ($n = 1000$) that included a question regarding their "interest in participating in a psychologic experiment" among other questions. The control group was recruited among those who responded positively to this question after the data from the alcoholic group had been collected. Of the 45 control participants, 11 were smokers. An effort was made to match control participants to the experimental group with regard to the age and gender.

Equipment and Physiologic Analysis

The experiment was conducted in a sound-isolated experimental chamber (Tegner; 3.5 × 3.5 × 2.5 m). Psychophysiologic responses were recorded using PSYLAB software and hardware. For the heart rate (HR) recordings, Beckman silver/silver-chloride electrodes (Ag–AgC1 8 mm) were used with Beckman Electrode Electrolyte. The HR electrodes were attached laterally between the first and second ribs, with a reference electrode below the sternum. The cardiovascular activities measured in this experiment were 1) average cardiac beat per minutes (bpm) sample (HR mean duration of R- spike to R-spike interval in msec and 2) the average of the absolute values of successive differences in R–R intervals in msec (mean of successive differences). The latter index of cardiac function is highly correlated with vagal sources of heart rate variability and is therefore labeled HRV in this study (Fox 1983; Yeh et al 1973).

Questionnaires

Participants were asked three questions on a 10-point Likert scale of self-reported perceived ability to resist a drink, urge to drink, and general fidgetiness (see Stormark et al 1995). The participants were asked the following: "How difficult would it be for you to resist a drink right now?" "How strong is your desire for a drink right now?" and "How fidgety do you feel right now?".

WHITE BEAR THOUGHT SUPPRESSION INVENTORY (WBSI). This scale measures the tendency to suppress thoughts and be bothered by unwanted and intrusive thoughts (Wegner and Zanakos 1994). It has been studied extensively and shows satisfactory psychometric stability. Each item is rated on a scale from 1 (totally disagree) to 5 (totally agree). High scores mean high levels of suppression and intrusion of unwanted thoughts.

The WBSI is related to various measures of psychopathology and dysfunctional psychologic states (Smari and Holmsteinsson 2001).

OBSESSIVE COMPULSIVE DRINKING SCALE (OCDS). The OCDS is a two-dimensional scale measuring obsessive thoughts of drinking and compulsive drinking behavior. The scale has two main subscales: the obsessive drinking subscale, which measures efforts and abilities to resist thoughts of alcohol and drinking, and the compulsive drinking scale, which measures the ability and effort to resist impulses to drink. Each item is anchored on a scale from 0 to 4, on which high scores indicate decreased control and low scores indicate increased control of alcohol-related cognitions and impulses. To avoid tautologic references and because of the in-treatment context, items seven and eight, which report amount of current drinking, were left out of the compulsive subscale (Schippers et al 1997). The OCDS is related to various other measures characterizing symptoms of chronic alcohol abuse and has in some studies been found to be related to treatment results (Anton et al 1995). This questionnaire was not administered to the control group.

SUBJECTIVE MOOD. Subjective mood was measured by the mood scale of Diener and Emmons (1985), a nine-item self-report instrument used to assess positive (four items) and negative (five items) mood anchored on a scale rated from 1 (not at all) to 7 (a lot). This measurement has been used successfully in other addiction studies and has been thoroughly validated to show the two most basic and independent aspects of mood (i.e., positive and negative affective state; Diener and Emmons 1985). We used a computerized dictionary to translate the positive and negative adjectives into Norwegian. Reliability item analysis within our sample suggested satisfactory psychometric stability for the positive (Cronbach's alpha = .89) and negative (Cronbach's alpha = .82) mood scales.

SEVERITY OF DEPENDENCE SCALE (SDS). The SDS is a four-item scale that assesses the level of psychologic dependence to addictive substances (alcohol in this study). Each question is rated on a 4-point scale (e.g., "Do you believe your use of alcohol is out of control?" rated as "never/almost never," "sometimes," "often," "always/almost always"). This instrument has been extensively validated and has been shown to have good psychometric properties in different samples of substance abusers (Gossop et al 1995).

CRAVING. Craving over the previous 3 weeks was measured using three questions on a 10-point scale rated from zero (very little) to nine (a lot) similar to traditional craving measures used in other studies to cross-validate new craving measures (Anton et al 1996; Schippers et al 1997).

Procedure

Regional Medical Ethical Committee for Western Norway approved this study. Before the experiment, participants were instructed to abstain from smoking and eating for 2 hours before the experiment was conducted. After giving informed consent to participate in the experiment, the participants filled out the questionnaires (OCDS, WBSI, SEDS). They were then presented with a picture identification task in which 26 masked slides of alcoholic and nonalcoholic content were briefly (20 msec) presented, and the viewer was asked to decide whether the pictures had alcoholic or nonalcoholic content. This data collection was related to another experiment and will therefore not be described here. Greater details about such forced choice tasks can be found elsewhere in the literature (e.g., Öhman and Soares 1994).

Participants filled out the mood measures and the questions related to fidgeting, the urge to drink, and perceived difficulty resisting a drink. It was then explained to the subjects that they were about to listen to an audiotape and that a female voice would tell them when to press a button on the table in front of them. This pushbutton triggered an event marker registered by the PSYLAB system to mark the onset and offset of physiologic data recording during the preimaginary exposure (introduction and rest period) and the imaginary alcohol exposure. Approximately 4 min passed between the picture identification task and the current experiment. The experimental chamber was then closed and the audiotape started, which began with a 1-min silence followed by a 1-min orientation about the procedures and a 3-min, 20-sec silence. At the end of the tape, the subject heard an imaginary alcohol script (1 min, 39 sec in length), a shortened and modified version of an imagery script that successfully increased the urge to drink in alcoholics in other studies; this script is described in greater detail elsewhere (Felstead et al 1994). During this task, the participants were instructed to vividly imagine different situations in which the use of alcohol would be expected to be experienced as tempting (for example, at party, in a bar, passing a liquor store, when in a depressed mood).

After the tape was finished, the experimenter went into the room and asked the participant to fill out the questionnaires concerning the urge to drink, fidgeting, and difficulty resisting a drink. The participants were informed that the session had ended, and the majority proceeded to another experiment not discussed in this article. After the data collection ended, all participants went through a debriefing process and were paid 200 Norwegian kroners (approximately $25).

Design and Statistical Analysis

The preimaginary exposure HR data were averaged across the 4-min, 33-sec preexposure period. The HR data during the exposure was averaged over the 1-min, 39-sec exposure recording. A two-way analysis of variance (ANOVA) was conducted, with the factors group (alcoholics and control) and exposure (preimaginary exposure and imaginary alcohol exposure) as independent variables and HR as the main dependent variable. To test the specific predictions suggested in the hypotheses, planned comparisons were conducted (Hays 1988). Because of a priori hypotheses, one-tailed tests were used to address the statistical significance of these contrasts. Tukey's honest significant difference post hoc tests were used to test the significance of interactive effects. Pearson's Product–Moment correlation analyses were used to test the association between HRV and the scores on the questionnaires (WBSI, OCDS, mood) addressed in the hypotheses.

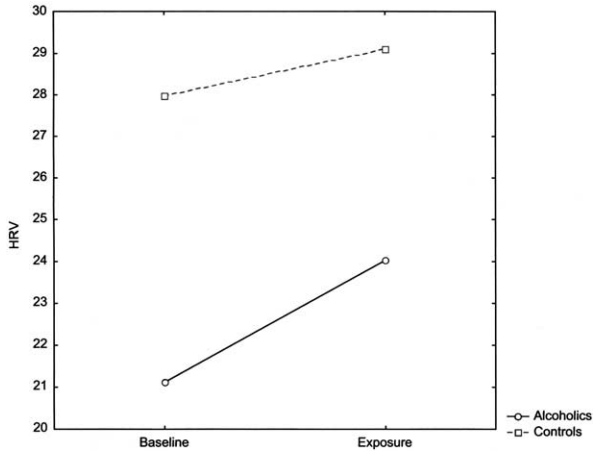

Figure 1. Mean heart rate variability (HRV; mean square of successive differences) in alcoholic and control participants at baseline and under exposure of alcohol script.

Results

Heart Rate Variability

There was a negative relationship between HRV and HR, supporting the theoretical assumption that HRV mainly reflects level of inhibitory effects of HR ($r = -.47$, $p < .0001$). As expected, the alcoholic participants had a generally lower HRV compared with the control group: $F(1,89) = 2.67$, $p = .05$ (one-tailed). Planned comparison analyses suggested that the alcoholic participants had significantly lower HRV compared with the control participants during the preimaginary exposure: $F(1,89) = 3.08$, $p = .04$ (one-tailed). This effect was not significant in the exposure period: $F(1,89) = 1.8$, $p = .18$. The alcoholic participants had a significant increase in HRV during the exposure: $F(1,89) = 2.66$, $p = .05$ (one-tailed). No such increase in HRV during exposure was found in the control group: $F(1,89) = .79$, $p = .37$ (see Figure 1). Because smoking was more common in the alcoholic compared with the control group, the previous analyses were repeated using smoking as a covariant (coded as smoking or not smoking). The results suggested that the covariate of smoking was not significant ($p = .45$), nor did adding this factor in the analysis change the results. A two-way ANOVA analysis found no significant differences in tonic HRV between alcoholic participants reporting use of medication and those not receiving such medication on preimaginary exposure or within the exposure condition.

Tonic Heart Rate

Alcoholics had significantly faster HR (78.75 bpm) than the control group (71.04 bpm): $F(1,90) = 10.07$, $p = .002$. The heart rate was also generally faster at preimaginary exposure (75.05 bpm) than during the imaginary exposure (74.6 bpm): $F(1,90) = 4.11$, $p = .046$. The interaction between group and exposure was not significant, $F(1,90) = 3.27$, $p = .07$; however, planned comparisons suggested that the alcoholics had significantly lower HR (78.48 bpm) during the exposure compared to preimaginary exposure (79.04 bpm): $F(1,90) = 7.86$, $p = .006$. No such effect was found within the control group: $F(1,90) = .002$; $p = .88$.

The Urge to Drink, Difficulty Resisting a Drink, and Fidgeting

Planned comparison analyses suggested that alcoholics reported a significant increase in the urge to drink, difficulty resisting a drink, and fidgeting after the imaginary exposure [$F(1,91) = 14{,}01$, $p = .0003$; $F(1,91) = 12.13$, $p = .0008$; and $F(1,91) = 17.75$, $p = .00006$, respectively] but no such increase was found in the control group. Conversely, the control subjects had a tendency to lower fidgeting rating after the imaginary alcohol exposure than at preimaginary exposure: $F(1,91) = 3.53$, $p = .07$.

Correlations between HRV and Self-Report Questionnaires

Correlation analyses between HRV and self-report variables revealed a significant negative correlation between the White Bear Thought Suppression Scale and HRV. This association was mainly found in the alcoholic group. There was also a significant negative correlation between negative mood and HRV and a positive correlation between positive mood and HRV in the whole sample (see Table 2); however, there was a significant negative correlation between self-reported compulsive drinking behavior (OCDS) and HRV during the exposure in the alcoholic group. Preimaginary exposure HRV activity was subtracted from HRV during the imaginary exposure to test if the association between HRV and the compulsive subscale was specific for the exposure condition. The results suggested that this association remained highly significant even after correcting for baseline levels in HRV: $r = -.41$, $p = .004$. Substantial differences existed between the alcoholic and control participants in level of educational background. To test whether this factor was related to tonic HRV and therefore could be an important confounding factor in the comparison between the groups, a correlation analysis was conducted testing the association between HRV at baseline and education. The results suggested that there was no significant association between these variables ($r = .09$, $p = .42$).

To explore the association between compulsive items of the OCDS and HRV a post hoc ANOVA analysis was conducted, dividing the alcoholics into two groups on the

Table 2. Correlation between Heart Rate Variability and White Bear Thought Suppression Inventory (WBSI), Obsessive Compulsive Drinking Scale (OCDS), and Positive and Negative Mood

	Alcoholics		Control Subjects		All Groups	
Measure	Preexposure	Exposure	Preexposure	Exposure	Preexposure	Exposure
WBSI	−.31[a]	−.31[a]	.09	−.01	−.21[a]	−.21[a]
Negative Mood	−.16	−.26	−.20	−.24	−.24[a]	−.29[a]
Positive Mood	.21	.23	−.01	.03	.21[a]	.21[a]
OCDS Obsessive	−.10	−.11				
OCDS Compulsive	−.10	−.31[a]				
OCDS Total	−.15	−.27				

[a] $p < .05$ two tailed test.

basis of their scores on the compulsive items of the OCDS. This median split division resulted in two groups that we labeled as "high and low compulsive drinking," those having a compulsive drinking score (CDS) lower than or equal to 11, with mean score of M = 6.7 (range: 0–11) were considered low compulsive. The total number of alcoholic participants in this group was 21. There were 25 individuals in the high compulsive alcoholic group, for whom the average compulsive score was M = 13.9 (range: 12–20). The result of the ANOVA analysis did not find any overall differences in HRV between high and low CDS groups [$F(1,44) = 1.25$, $p = .27$]. As in the earlier analyses, a significant increase in HRV was found in both alcoholic groups after exposure to imaginary alcohol stimuli [$F(1,44) = 6.08$, $p = .0177$]. The interaction between the two CDS groups and condition (preimaginary exposure vs. imaginary exposure) was significant: $F(1,44) = 13.70$, $p = .0006$. This effect was explored by conducting a planned comparison analysis within the two groups. The low CDS group showed a significant increase (see Figure 2) in HRV after imaginary exposure [$F(1,44) = 17.48$, $p < .0001$], whereas there was no change for the "high" CDS group [$F(1,44) = .83$, $p = .36$].

Further analyses were conducted on the self-report measures to investigate whether the high CDS group reported more difficulties resisting alcohol after the imaginary alcohol script compared with the low CDS group. Whereas the high CDS group reported significantly increased difficulty resisting a drink after imaginary exposure [$F(1,45) = 8.77$, $p = .0048$], no such increase was found in the low CDS group [$F(1,45) = .47$, $p = .49$; see Figure 3].

Discussion

The main results of our study may be summed as follows. First, as expected, alcoholic participants had lower HRV compared with the nonalcoholic control group. Second, the imaginary alcohol exposure increased HRV in the alcoholic participants. Third, across the groups, an inverse association was found between HRV and negative mood and a positive association between positive mood and

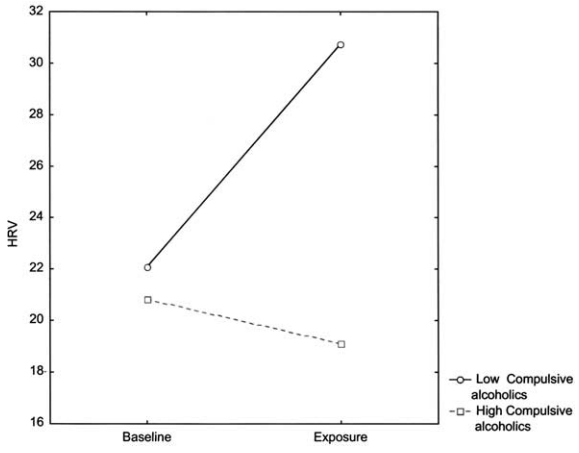

Figure 2. Changes in heart rate variability (HRV) in "high" and "low" compulsive alcoholic participants after exposure to alcohol cues.

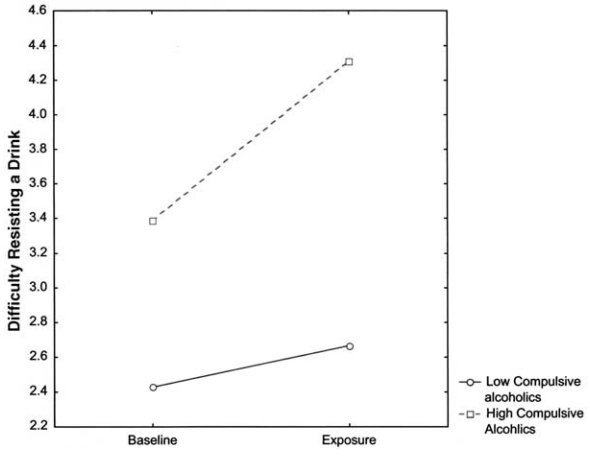

Figure 3. Mean difficulty resisting a drink in "high" and "low" compulsive alcoholic participants after exposure to alcohol script.

HRV. Fourth, HRV was negatively correlated with compulsive drinking during the imaginary alcohol exposure in the alcoholic participants. Fifth, within the alcoholic group, HRV was negatively associated with chronic thought suppression (WBSI).

Generally, these findings are in agreement with the neurovisceral integration model and the polyvagal theory that suggests HRV is a marker of the level of cognitive, behavioral, and emotional regulatory abilities (Thayer and Lane 2000). In previous studies, low HRV has been associated with various psychiatric conditions such as depression, panic disorder, posttraumatic stress disorder, and generalized anxiety disorder (Thayer and Lane 2000). The fact that the alcoholic group had generally lower tonic HRV compared with the nonalcoholic control group indicates that such reduced HRV may also be a factor in alcohol abuse; however, such group differences in HRV provide only indirect support for the theory that low HRV in alcoholics may be related to impaired inhibitory mechanisms as would be predicted by the neurovisceral integration model. Low HRV may, for example, be caused primarily by somatic complications related to alcohol abuse and withdrawal that are perhaps unrelated to inhibitory mechanisms and affective control. In our study, however, we assessed individual differences in mood, thought control, and compulsive drinking and thus were able to explore the relationship between HRV and these psychologic processes.

As predicted by the theory, low HRV was inversely associated with negative affect and positively associated with positive affect. Therefore, these two main dimensions of mood inversely mirrored each other in the hypothesized direction in relation to tonic HRV. These results indicate that increased vagal influences on HR are more apparent in positive mood states, whereas negative mood states are characterized by a relative sympathetic nervous system activation that served to minimize the effects of vagal control on HR. These findings therefore are consistent with other research linking low HRV to dysphoric or negative affective states and high HRV to more positive affective states characterized by flexible attentional focus and an openness to social engagement (Friedman and Thayer 1998a; Hughes and Stoney 2000; Porges 1995; Porges et al 1996; Thayer et al 1996); however, these associations between affect and HRV were found across the two experimental groups and thus did not reveal specific sources of problems in affective regulation within the alcoholic group.

To investigate vagal activity associated with the processing of alcohol-related information, participants were exposed to an "imaginary alcohol script." We found a corresponding increase in HRV in alcoholics after exposure to the imaginary alcohol script as has been reported in recent studies that applied a traditional olfactory alcohol cue exposure paradigm (Jansma et al 2000; Rajan et al 1998). Thus, our study extends previous findings by replicating their results using an imaginary alcohol script to prime alcohol-related cognition (Felstead et al 1994).

Respondent conditioning theories predicting that responses to drug-related cues either reflect aversive abstinence symptoms (Wikler 1965) or mimic drug effects (Eikelboom and Stewart 1982) have dominated explanatory models in cue reactivity studies during the last decade. Because withdrawal symptoms are characterized by autonomic hyperactivity (increased sympathetic activity) and thus removal of vagal tone, it is difficult to explain the increase in HRV under the imaginary alcohol script as conditioned abstinence (American Psychiatric Association 1994; Wikler 1965). The increase in vagal activity during the exposure also cannot be explained as a reflection of the pharmacologic effect of alcohol because the acute effect of alcohol results in a sharp reduction of vagal tone (Eikelboom and Stewart 1982; Weise et al 1986). The failure of these conditioning models to explain our findings is consistent with the increasing concern in the literature about the applicably of such "one-dimensional" conditioning models explaining the results of cue reactivity studies (Glauter and Remington 1995; Laberg 1990; Tiffany 1995).

As an alternative to such passive respondent models, some researchers have advocated the use of information-processing theory to understand how dependent individuals react in their encounter with "drug-related" cues (Tiffany 1990, 1995). Because HRV is related to activity in frontal brain areas involved in cognition and impulse control (Thayer and Lane 2000), we speculated that tonic HRV would be an index of nonautomatic inhibitory processes aimed at suppressing and controlling automatic drug-related cognitions. To test this hypothesis more directly, the association between HRV and problems with controlling drinking-related impulses were studied.

Consistent with this hypothesis, the compulsive subscale of the OCDS was found to be inversely associated with HRV in the alcohol-exposure condition, thus suggesting that HRV may be an indirect indicator of the level of impulse control associated with drinking. These findings are therefore consistent with Stormark et al (1998), who found that sustained HR acceleration (lack of vagal inhibition) when processing alcohol-related information was related to compulsive drinking and "locked-in attention." Post hoc analysis further suggested that alcoholics who expressed a relatively high ability to resist impulses to drink (OCDS) had the clearest increase in HRV under the alcohol exposure. Interestingly, this "low-compulsive" group did not report any difficulties resisting alcohol after "the imaginary alcohol exposure." Because of the explor-

atory nature of these analyses, care should be taken in generalizing from this finding; however, although preliminary, this study suggests that alcoholics may actively inhibit or compensate for their involuntary attraction to alcohol-related information by activation of higher nonautomatic cognitive processes (Tiffany 1995). Such conscious avoidance has previously been demonstrated in studies on attentional processes in alcoholics (Stormark et al 1997) and by the fact that frontal brain structures involved in inhibition and control of affective information are often highly activated in the processing of alcohol-related cues (Anton 1999). Furthermore, this interpretation is in agreement with other studies suggesting that high HRV during challenging tasks is associated with recovery from acute stress disorders (Sahr et al 2001) and successful recovery from anxiety and depressive disorders (Balogh et al 1993; Middelton and Ashby 1995).

Several studies have indicated that low HRV is associated with impaired cognitive control and perseverative thinking (Thayer and Lane 2002). Consistent with these reports a negative association was found between HRV and chronic thought suppression. The WBSI assesses efforts to eliminate thoughts from awareness while experiencing frequent intrusions of such "forbidden" thoughts and thus represents an interesting and well-validated measure of ineffective thought control (Wegner and Zanakos 1994). Thought suppression has been found to be an especially counterproductive strategy for coping with urges and craving (Palfai et al 1997a, 1997b) and may even play a causal role in maintaining various clinical disorders (Wenzlaff and Wegner 2000). To our knowledge, this is the first time a link between physiologic indicators of a lack of cognitive flexibility (low HRV) and chronic thought suppression has been demonstrated. Thayer and Friedman (2002) have reviewed evidence indicating that there is an association between vagally mediated HRV and the inhibitory role of the prefrontal cortex. Consistent with Thayer and Lane (2000), this study suggests that impaired inhibitory processes are significantly related to ineffective thought control. The fact that this association between HRV and WBSI was only found in the alcoholics may be related to the fact that only this clinical group shows signs of such faulty thought control.

Wegner and Zanakos (1994) suggested that thought suppression is particularly ineffective when the strategic resources involved in intentional suppression are inhibited or blocked (Wegner 1994). Consistent with this hypothesis, our findings show that those reporting high scores on WBSI show signs of impaired inhibitory functioning as indexed by low vagally mediated HRV. Our study may serve as an example of a more integrative approach to understanding the often dire consequences of faulty cognitive control based on theories of ineffective thought control strategies (Wegner 1994; Wenzlaff and Wegner 2000) and studies of the biological bases for such cognition (Thayer and Lane 2000, 2002). It is hoped that such an approach may resolve some of the theoretical and empirical controversies that affect this area of research (Purdon 1999; Smari 2001).

Some limitations of our study need to be addressed. First, basic differences in vagal activity may be related to factors other than the specific problem of alcohol abuse. A substantial number of the participants were under some form of medication that could hypothetically affect their responses. Future studies should pay more attention to the effect of medication on alcoholics' autonomic activity in simulated high-risk situations (Carter and Tiffany 1999). Other confounding variables, such as differences between the groups in sociodemographic background, psychopathology, and intellectual ability, may be associated with HRV independent of the problem of alcoholism. The result of post hoc analyses suggested that when factors such as smoking and education were controlled, the basic findings of this study were not altered. It is difficult to control for other variables that may naturally covary with alcohol dependency, such as the existence of other psychopathologic disorders (Tomasson and Vaglum 1995). Importantly, however, we do not expect that impaired inhibitory functioning is specific to alcohol abuse but may be a risk factor in many other clinical conditions (Thayer and Lane 2000). Controlling for such factors may therefore seem arbitrary and theoretically unjustified (Miller and Chapman 2001). Research on brain structures involved in emotion regulation show that such functions may be impaired in depressive patients (Bremner et al 2002). Thus, the possibility cannot be excluded that the association between HRV and chronic thought suppression is mediated by dysphoria. Studying specific autonomic functions related to alcoholics' coping in high-risk situations therefore remains an important challenge of future research.

In conclusion, our study suggests that HRV is a sensitive indicator of cognitive and emotional regulation. More specifically, the problem of alcohol abuse may be partially related to such defective inhibitory mechanisms, a conclusion shared by other researchers (Anton 1999; Lyvers 2000). Studies on depressed patients suggest that treatment can increase HRV and that such an increase can be highly related to successful recovery after treatment (Thayer and Lane 2000). Similarly, our study suggests that such inhibitory mechanisms may play an important role in successful relapse prevention (Marlatt and Gordon 1985).

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