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Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder

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ABSTRACT

Objective: Stress plays a role in the pathology of bulimia nervosa and binge eating disorders, but it is unclear whether they involve similar disturbances of biological stress responses.
Patients and methods: We recruited 25 patients with binge eating behavior, 12 with bulimia nervosa (BN) and 13 with binge eating disorder (BED), and compared them with 13 obese non-binge eaters (NBED). We measured heart rate variability in response to mental stress tasks, and concentrations of leptin, glucose and insulin in the blood.
Results: Heart rate stress reactivity was highest in BN patients. Heart rate variability did not change during mental stress in BN and BED patients, but reduced as expected in the NBED group. During post-stress recovery, heart rate variability decreased in BN, was maintained in BED and increased as expected only in the NBED group.
Conclusions: BN and BED patients exhibit limitations in autonomic stress reactivity and recovery capacity.

1. Introduction

Bulimia nervosa (BN) and binge eating disorder (BED) are both characterized by recurrent episodes of binge eating, but differ in compensatory behavior. Bulimic patients regularly use self-induced vomiting or misuse laxatives to control their weight, whereas individuals with binge eating disorder usually suffer from uncontrolled weight gain and obesity (Munsch and Beglinger, 2005).

It is thought that stress plays an important role in the pathology of both disorders (Pinaquy et al., 2003; Koo-Loeb et al., 1998). Heightened negative mood has frequently been described as a precursor to binge eating episodes in BN and BED (Levine and Marcus, 1997; Eldredge and Agras, 1996; Telch and Agras, 1996) as well as obesity. Individuals with BN and BED experience greater negative mood before binge eating episodes and perceive challenging situations as more stressful compared with healthy individuals (Pinaquy et al., 2003; Wolff et al., 2000; Hansel and Wittrock, 1997; Kjelsas et al., 2004). Additionally, several studies have shown an increase in hunger and desire to binge following experimental stress tasks (Cattanach et al., 1988; Gluck et al., 2004; Tuschen-Caffier and Vogele, 1999). Therefore, understanding the physiological mechanisms underlying stress responses would be useful for the identification of physiological correlates of binge eating patterns in BN and BED disorders.

There is evidence that people with BN and BED differ in autonomic functioning from healthy individuals. Patients with BN exhibit blunted sympathetic activation in response to mental stress (Koo-Loeb et al., 1998) and reduced 24 h blood pressure (Cong et al., 2004). Furthermore, results of a comparison of BED and controls suggested higher stress vulnerability in the BED population (Friederich et al., 2006). However, obesity might confound these results, as other studies on obese subjects have shown a change of stress response profiles due to obesity itself (Laederach-Hofmann et al., 2000; Valensi et al., 1995).

Similarities in biological stress vulnerability could be a key to understanding binge eating behavior in BN and BED. But studies of autonomic stress responses in BN and BED individuals are rather scarce. The purpose of this study was to assess the stress responses of patients with similar binge eating behavior but different diagnoses. In order to investigate the capacity of the autonomic nervous system to adapt to stressful conditions, we measured heart rate variability (HRV) and assessed autonomic activity during mental stress testing. We hypothesized that BN and BED patients would show similar disturbances of autonomic stress responses in comparison with non-binge eaters (NBED).

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2. Materials and methods

2.1. Participants and study protocol

Thirty-eight women agreed to participate. Bulimia nervosa (BN) was diagnosed in 12 participants, binge eating disorder (BED) in 13 and obesity without binge eating behavior (NBED) in 13 participants, according to DSM IV diagnostic criteria (Amercian Psychological Association, 1994). Individuals had to show stabilized electrolyte conditions, unchanged weight levels during the past 10 to 12 weeks and had to undergo a routine physical examination and diagnostic interview conducted by an experienced psychiatrist before being asked to participate in the study. Individuals with a medical history of cardiovascular disease, metabolic disease, other disease or medication that could influence autonomic functioning were excluded. All participants were recruited consecutively from the psychiatric outpatient clinic at the University Hospital of Berne and from a private clinic for eating disorders 'Klinik Wysshoelzli', both in Switzerland. All participants were referred to these specialized units by their family doctors, and were asked to participate only if the inclusion criteria for the study were fulfilled. Participants gave written informed consent to the study procedures which were approved by the local Research Ethics Committee.

2.2. Questionnaires

The German version of Stunkard and Messick's Three-Factor Eating Questionnaire (Pudel and Westenhoefer, 1989; Stunkard and Messick, 1985) was used to measure the three scales 'cognitive restraint', a tendency to control food intake in order to prevent weight gain; 'disinhibition' a term describing a combination of emotional and external eating, and 'hunger'. Additionally, symptoms of eating disorder such as fear of weight gain, dissatisfaction with one's own figure, binge behavior, nausea and vomiting after eating, feelings of external eating pressures and excessive demands, perfectionism, interpersonal reservations and fear of one's own feelings were assessed by the self-report Eating Disorder Inventory (Diehl and Staufenbiel, 1994). Finally, the German version of Hospital Anxiety and Depression Scale (Herrmann, 1997; Zigmond and Snaith, 1983) was administered to assess symptoms of depression and anxiety.

2.3. Cardiovascular assessment and stress testing

Autonomic function testing was performed in 3 conditions: at rest, during mental stress, and recovery from mental stress. Each condition lasted 10 minutes according to the guidelines of the joint European/North American task force (1996) on heart rate variability. All testing sessions were carried out between 08:00 am and 12:00 pm. Patients were required not to do any physical activity during the 24 h before the testing session, not to drink caffeine-containing liquids, and were tested in a fasting state (following overnight fast). All participants were tested in their first week after menstruation only (Ettinger et al., 1998; Matsumoto et al., 2007). The laboratory had no natural light and was heated to room temperature (18–20 °C). After fitting of measuring devices an equilibration period of 20 minutes took place. The mental stress test involved administration of the Stroop color/word interference task using standardized methods (Hoshikawa and Yamamoto, 1997). The task difficulty was titrated on the basis of the individual's performance by testing system 'Wiener Testsystem' (Schuhfried GmbH, Austria).

Heart rate, blood pressure, and respiration were recorded simultaneously with a computer-based system (Task Force Monitor ®, CN-Systems, Graz, Austria). The Einthoven lead II of the electrocardiogram was used to detect the R-peaks of the electrocardiogram (ECG). Blood pressure was measured non-invasively and continuously from the fingers (Habenbacher et al., 2002). The continuous blood pressure signal was equilibrated in five minute intervals by oscillometric blood pressure determination in the contralateral arm. Respiration frequency was recorded using impedance pneumography and stroke volume was estimated using impedance cardiography. The ECG and beat-to-beat blood pressure signals were sampled with a frequency of 1000 Hz and changes of thoracic impedance due to respiration at 50 Hz.

HRV was assessed using spectral analysis. Previous analyses have shown that the commercial algorithm used to prepare heart period data for spectral analysis (TFM Software V2.4, CN-Systems, Graz, Austria) fails to detect all artifacts. After applying this method, we additionally visually displayed heart period data to screen out any residual outliers and artifacts. Using this conservative procedure, fewer than 10% of the total data were problematic and had to be excluded. ECG sequences that showed premature beats or other artifacts were also excluded from the analysis. A commercial software package was then employed to determine HRV with an adapted autoregressive model as proposed by Bianchi et al. (1997), using a recursive least squares algorithm.

Power spectral densities (in ms^2) were quantified by the area within the frequency bands which were determined according to international guidelines (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). We calculated total power (HRV-TP) in the range 0.003–0.4 Hz; low-frequency (LF) power, range 0.04–0.15 Hz; and high-frequency (HF) power, range 0.15–0.4 Hz. Data were skewed, so all spectral values were normalized by natural logarithms (ln) (Malliani et al., 1991). HRV-TP is an index of total heart rate variability (HRV). LF HF ratio reflects sympathetic/parasympathetic balance, with higher values indicating greater sympathetic control (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). By contrast, the HF power component is a marker of parasympathetic influence on cardiac chronotropy. Baroreflex sensitivity was calculated by using hemodynamic data assessed with impedance cardiography (ICG) using the dZ/dt paradigm which is part of the TFM program.

2.4. Blood samples

Blood samples for hormone assays and glucose were obtained fasting after the heart rate variability testing session between 8:00 and 12:00 h in all participants. Glucose was assessed by using hexokinase enzyme assay (Roche, Modular P800, Roche Diagnostics Switzerland) and insulin by microparticle-enzyme immunoassay (MEIA), Abbott Axsym. Plasma leptin concentrations were determined by a LEP-R40 radioimmunoassay kit purchased from Mediagnost (Reutlingen, Germany). Sensitivity of the method was 0.04 ng/ml. The homeostasis model assessment index (HOMA-R) of insulin resistance was calculated (Gutt et al., 2000).

2.5. Statistics

Basic descriptive statistics and repeated measures analysis of variance were used to analyze the data, with trial (rest, mental stress, and recovery) as the within-subject factor and patient group (BN, BED and NBED) as the between-subject factor. Analyses were performed with a PC-based SPSS 14.0 (SPSS Inc. Chicago, Illinois, USA). Data are reported as means ± standard deviations (SD). A p-value of less than 0.05 was considered significant.

3. Results

3.1. Participant characteristics

Participants differed in age and BMI (Table 1). *Post hoc* tests indicated that the BN group was younger on average than the other