



Confrontation with blood and disgust stimuli precipitates respiratory dysregulation in blood–injection–injury phobia

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ABSTRACT

Blood–injection–injury (BII) phobia patients sometimes faint during exposure to relevant stimuli. However, mechanisms and timing of physiological adjustments in BII phobia remain poorly understood. In a larger sample of 60 patients and 20 controls, we sought to replicate findings of a prior study demonstrating the role of hyperventilation in the phobic response. We also investigated the timing of respiratory adjustment across an extended exposure recovery period. In addition, because intense disgust is commonly reported by patients, responses to surgery films were compared to a pure disgust film. End-tidal PCO₂ dropped significantly while volume and flow increased during the surgery film in patients compared to controls and to other emotional films except disgust. Patients recovered quickly following the disgust film but not the surgery film. PCO₂, volume, and flow parameters showed robust associations with anxiety, disgust, and physical symptoms. Findings suggest that respiratory adjustments during and after phobic exposure may provide a critical missing link in the understanding of the psychophysiology of this singular disorder, including why fainting often occurs after the stimulus is removed.

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Blood–injection–injury (BII) phobia is a common and often debilitating phobia with profound consequences for many of its sufferers. Unlike any other anxiety disorder, BII phobia often involves a syncopal response, with upwards of 75% of individuals with this phobia reporting a history of fainting or near fainting at the sight or even discussion of blood, injuries, or needles (American Psychiatric Association, 2000). The BII-phobia response is described in the literature as a vasovagal syncope, characterized by bradycardia and/or hypotension leading to critical reductions in cerebral blood flow and ultimately fainting (e.g., Graham et al., 1961).

To date, cardiovascular parameters have been the main focus of psychophysiological research on BII phobia (e.g., Öst et al., 1989). However, studies are still equivocal with respect to the role of autonomic adjustments, such as parasympathetic activity, in the disorder (e.g., Friedman et al., 1993; Sarlo et al., 2008). Research investigating neurocardiogenic syncope (also sometimes referred to as *emotional fainting*, such as in BII phobia) suggests that response patterns may be more complex in that peripheral cardiovascular changes do not always directly precede the fainting-related reductions in cerebral blood flow (e.g., Grubb et al., 1998).

Thus, other factors may play a role in the development or exacerbation of an emotionally triggered syncopal event, includ-

ing hyperventilation (Foulds, 1993). Indeed, changes in respiration leading to hypocapnia have been demonstrated prior to the onset of syncope during tilt-table testing (e.g., Lipsitz et al., 1997) and orthostatic intolerance (Novak et al., 1998) in individuals with and without drops in heart rate (HR) or blood pressure (BP). Furthermore, sensitivity to drops in PCO₂ leading to more pronounced vascular changes in the brain and skeletal muscles has been found in individuals with neurocardiogenic syncope (Norcliffe-Kaufmann et al., 2008).

Although respiration has received some attention in research on neurocardiogenic syncope, little focus has been directed to its potential contribution to the BII-phobia-specific response pattern. BII-phobia studies that have included respiration measurements have typically restricted their focus to respiration rate (RR), with little evidence emerging for a role of RR in the disorder (e.g., Vögele et al., 2003). However, in a recent study that also measured tidal volume (V_T , total volume of air moved in and out of the lungs in one breath) and partial pressure of carbon dioxide (PCO₂), substantial drops in PCO₂ occurred when BII-phobia patients were exposed to phobia-relevant film material (Ritz et al., 2005), indicating that patients reached hypocapnic levels during these periods. In addition, an interesting pattern of respiratory changes was observed: while RR remained largely unchanged, pronounced increases in V_T and minute ventilation (V'_E , the total amount of air that is exhaled within 1 min) were observed (Ritz et al., 2009). These increases correlated positively with increases in reports of lightheadedness and dizziness.

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Thus, in this sample of BII-phobia patients, hyperventilation was a result of *deeper* rather than faster breathing. This phenomenon appears to be a critical physiological adaptation to feared stimuli, which may also produce symptoms typical for fainting. Hyperventilation, which is common to the fear response seen in anxiety disorders, is known to reduce cerebral blood flow by constricting blood vessels in the brain (Fried, 1993). Thus, for individuals with a propensity to faint at the sight of BII stimuli, reductions in PCO₂ might potentiate this response and increase the likelihood that the individual will faint in the anxiety-provoking situation.

Although the results of this previous study (Ritz et al., 2005, 2009) were intriguing, generalization was limited due to the small sample size. Therefore, we sought to replicate these findings in a larger patient sample. In addition, clinical observations and research have suggested that the recovery period from exposure to phobia-relevant material might be particularly problematic for some BII-phobia patients, with fainting sometimes occurring after the BII stimulus is removed (e.g., Graham et al., 1961). Indeed, Öst et al. (1984) found that for some BII patients, critical drops in BP and/or HR were evident up to 4 min past withdrawal of the BII stimulus. Ritz et al. (2005) observed that one BII patient (out of a total of 12) showed symptoms of faintness only after the stimulus was removed (after the 1-min recovery period). This patient presented with high levels of anxiety and low levels of PCO₂ (minima dropped to 25.1 mmHg) during the film and recovery. Because prior research has not systematically analyzed recovery phases, especially with respect to breathing pattern, an in-depth analysis of respiratory changes during recovery from exposure to BII-relevant stimuli was conducted in the present study.

Finally, because disgust has been hypothesized to play a central role in BII phobia, we sought to examine the specific role of disgust during exposure to BII-relevant as compared to disgust-relevant stimuli. Individuals with BII phobia have been found to report high levels of disgust within the phobia-relevant context (e.g., Tolin et al., 1997), with disgust sometimes reported as the predominant emotional response to blood and injury (but not injection) stimuli (e.g., Olatunji et al., 2007). In addition, some studies have found that individuals with BII fears report a general sensitivity to disgust across a range of disgust-related situations, such as scenarios involving rotting food (Sawchuck et al., 2000), although findings have not been consistent (e.g., Gerlach et al., 2006; Koch et al., 2002; Schienle et al., 2003).

Overall, recent findings suggest that disgust is an important avenue for investigation given that a complex interrelation between fear and disgust is likely present in BII phobia (e.g., Olatunji et al., 2007). Furthermore, the relationship between respiration and disgust in the response to BII stimuli remains largely unexplored. One exception is the study by Ritz et al. (2005) that examined respiratory dysfunction in BII phobia and found strong disgust responses during exposure to surgery films. These responses were associated with pronounced decreases in PCO₂ (Ritz et al., 2005). However, the psychophysiological response to BII-unrelated disgust stimuli has not been fully explored in this patient population. Therefore, in the current study we sought to investigate whether the presentation of disgust stimuli, independent from BII stimuli, would elicit distinct symptoms and respiratory adaptations similar to BII-relevant material in BII-phobia patients. This avenue of research could be particularly promising for obtaining a better understanding of fainting behavior and disgust sensitivity in this population.

1. Methods

1.1. Participants

We recruited 60 participants with BII phobia (81.7% female; mean age 27.9 years, SD = 9.0, range 18–49 years) and 20 gender- and age-matched, healthy controls (85% female; mean age 27.2 years, SD = 9.2, range 18–46 years) to participate in a study

investigating the role of respiration in the pathophysiology and treatment of BII phobia (data on the subsequent treatment component of the study will be the subject of a separate report).

Participants were recruited through online advertisement services and flyers posted around the local university campus. Individuals were identified through a phone screen as potential candidates and invited for an initial screening interview to determine eligibility. DSM-IV diagnosis was based on the Patient Version of the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV), Axis I Disorders Module (SCID-I, First et al., 1994). The interviews were conducted by an instrument-trained doctoral student.

General psychological exclusion criteria included a history of Bipolar I, current alcohol/substance abuse or dependence, or psychosis. Additional exclusion criteria included a history of stroke, cardiac disease, or lung disease as well as current diabetes, hypertension, hypercholesterolemia, and pregnancy. We also excluded current smokers because of the known influences of tobacco smoking on the airways and the autonomic regulation. Healthy controls were included only if they did not meet the criteria for any current or past history of specific phobia–BII type. In addition, healthy controls were excluded if they endorsed any current symptoms of psychiatric disorder. To help insure adequate differentiation between the patient and control samples, individuals with mild BII-related anxiety (i.e., more than very mild anxiety that is typical when getting an injection) were excluded.

BII patients were single (68.3%), married (20%), living with a partner (5%), separated or divorced (5%), or they chose not to respond (1.7%). Sixty-five percent identified themselves as White, 13.3% as Asian or Pacific Islander, and 15% as African American. For ethnicity, 7% identified themselves as Hispanic. Sixty-five percent were currently employed. All BII patients met DSM-IV criteria for Specific Phobia–BII type. Forty-seven percent met criteria for a comorbid disorder, including other specific phobia subtypes, panic disorder, obsessive–compulsive disorder, generalized anxiety disorder, mild major depressive disorder, binge eating disorder, and body dysmorphic disorder.

A history of fainting (including loss of consciousness) in situations related to blood, injury, or needles was reported by 27 (45%) BII patients. An additional 14 (23.3%) BII patients reported coming close to fainting in BII situations but were able to circumvent the syncopal response by using various “coping skills” such as lying down or taking deep breaths. All BII patients reported experiencing physical symptoms during exposure to BII stimuli (e.g., shortness of breath, hot flashes). The study was approved by the University IRB Committee. In accordance to ethical guidelines, informed consent was obtained from all participants. Eligible participants were paid \$25 for the session.

1.2. Experimental design

The one-session experimental design included presentation of short film clips to elicit six different emotional states: sadness (woman mourning over the death of her boyfriend; 300 s), happiness (British comedian driving into a truck full of mattresses; 272 s), anger (group of bullies picking on others; 302 s), disgust (contestants transferring ground-up insects using the mouth; 304 s), emotionally neutral (screensaver; 182 s), and fearfulness (bone graft surgery educational video; 291 s). Films were selected from commercially available media, medical education films (Ritz et al., 2005), and libraries of pre-evaluated emotion-inducing clips (Gross and Levenson, 1995). The phobia-related surgery film included a scene with surgeons making two large incisions in the leg, drilling a channel through the bone, and then extracting and harvesting bone marrow. Special attention was paid to choosing a disgust film that did not include any stimuli relevant to BII such as the illustration of blood or injury. For this film, contestants were sibling pairs competing in a disgust-oriented relay race. The first sibling would transfer live insects (e.g., worms and cockroaches) from a large container of bugs to a grinder using only his or her mouth, with the second sibling then catching the ground-up insects in his or her mouth and transferring them to a second container. The order of the films was randomized within the session and all films were followed by a recovery period (5 min for the surgery film and 1 min for all other films).

1.3. Measures

1.3.1. Physiological measurements

End-tidal PCO₂ was measured using a Capnocount Mini infrared, side-stream capnometer (Weinmann Inc., Mannheim, Germany). Participants wore a nasal cannula (Salter Labs; tube length, 7 ft) throughout the session, which sampled the exhaled breath at a rate of 150 ml/min. Participants were instructed to breathe through the nose with the mouth closed during assessments to ensure accurate sampling of the breath. Because of equipment failure, PCO₂ was monitored with another capnometer (Tidal Wave, Respirationics, Philips Healthcare, Andover, MA) for 12 participants. Both capnometers meet international accuracy standards (Biedler et al., 2003) and no systematic baseline differences were found between the two devices. PCO₂ values were stored along with the time and date of the measurement in the internal memory of the device.

Respiratory volume, stability, flow, and timing parameters were continuously monitored via respiratory inductance plethysmography (LifeShirt System; Vivosmetrics, Ventura, CA) throughout the session. Participants wore a lightweight vest with integrated inductive plethysmographic bands around the thorax and abdomen,

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