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The effects of sighing on the cardiovascular system

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ABSTRACT

Elicitation of high-amplitude oscillations in the cardiovascular system may serve to dampen psychophysiological reactivity to emotional and cognitive loading. Prior work has used paced breathing to impose clinically valuable high-amplitude ~0.1 Hz oscillations. In this study, we investigated whether rhythmical sighing could likewise produce high-amplitude cardiovascular oscillations in the very low frequency range (0.003–0.05 Hz). ECG, respiration, skin conductance, and beat-to-beat blood pressure were collected in 24 healthy participants during baseline, 0.1 Hz paced breathing, and 0.02 Hz paced sighing (1 sigh every 50 s, with normal breathing interspersed). Results showed that each sigh elicited a strong, well-defined reaction in the cardiovascular system. This reaction did not habituate when participants repeatedly sighed for 8.5 min. The result was a high-amplitude 0.02 Hz oscillation in multiple cardiovascular parameters. Thus, paced sighing is a reliable method for imposing very low frequency oscillations in the cardiovascular system, which has research and clinical implications that warrant further study.

1. Introduction

The influence of respiration on the cardiovascular system is well-established. The phenomenon of respiratory sinus arrhythmia has been extensively studied (e.g., Berntson, Cacioppo, & Quigley, 1993; Grossman & Taylor, 2007; Ritz, 2009). The phenomenon of heart rate (HR) baroreflex resonance in the cardiovascular system, which is observed as high-amplitude low-frequency oscillations in heart rate, blood pressure, and vascular tone when breathing is paced at a frequency of ~0.1 Hz (6 breaths per minute), further illustrates the impact of respiration on the cardiovascular system (Vashchillo, Zingerman, Konstantinov, & Menitsky, 1983; Vaschillo, Vaschillo, Buckman, Pandina, & Bates, 2011). Both of these respiratory phenomena are essential sources of variability in HR, vascular tone, stroke volume, and blood pressure (Grossman & Taylor, 2007; Vaschillo et al., 2011).

Greater cardiovascular variability is associated with better physical and emotional health (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). Moreover, amplifying cardiovascular variability through therapeutic techniques such as HR variability (HRV) biofeedback, which trains people over the course of several weeks to rhythmically breathe at 0.1 Hz (6 breaths per minute), can improve symptoms of asthma (Lehrer et al., 2004), fibromyalgia (Hassett et al., 2007), post traumatic stress disorder (Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009), major depression (Karavidas et al., 2007), neurosis (Chernigovskaia, Vashchillo, Petrash, & Rusanovskiĭ, 1990), and hypertension (McCraty, Atkinson, & Tomasino, 2003). Although the physiological mechanisms underlying the therapeutic effects of HRV biofeedback continue to be explored, enhanced sensitivity of the baroreflex (i.e., the autonomic reflex that links dynamic cardiovascular processes to each other and to neural processing) has been implicated (Chernigovskaia et al., 1990; Lehrer, Vaschillo, & Vaschillo, 2000). This theory suggests that the 0.1 Hz breathing element of HRV biofeedback "exercises" autonomic reflexes to increase cardiovascular variability and improve baroreflex functioning, much in the same way that exercising somatic muscles improves neuromuscular reflexes (i.e., coordination and balance). In a sample of healthy individuals, improvements in respiration (i.e., peak flow) and baroreflex gain were noted after several weeks of consistent HRV biofeedback training (Lehrer et al., 2003). This long-term improvement in baroreflex gain was not replicated

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(Lehrer et al., 2004) in a sample of asthma patients who showed no cumulative improvements in baroreflex gain, although their clinical symptoms improved and baroreflex gain was enhanced during HRV biofeedback suggesting that 0.1 Hz paced breathing, when properly performed, has in-the-moment effects on the baroreflex.

In this study, we sought to build on knowledge of how respiration influences cardiovascular variability by examining whether high-amplitude cardiovascular oscillations also could be elicited by using rhythmical sighing paced in the very low frequency (VLF) range (0.003–0.05 Hz). Prior studies (Hammer & Saul, 2005; van de Vooren et al., 2007; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002; Vaschillo et al., 2011) suggest that the vascular tone branch of the baroreflex is active in the VLF range and, therefore, a vascular tone baroreflex-specific resonance frequency may be observed in this range. Theoretically, triggering resonance in this range could be beneficial for mental and physical health in much the same way as are HR baroreflex 0.1 Hz resonance responses in the low frequency range. However, a major challenge to demonstrating resonance properties in the VLF range is that while most individuals, regardless of health status, can be trained to breathe at 0.1 Hz to trigger HR baroreflex resonance, few individuals other than monks or yogis (Lehrer, Sasaki, & Saito, 1999) can sustain a breathing rate in the VLF range. We present results of a study that trained young healthy adults to rhythmically sigh at 0.02 Hz (1 sigh every 50 s, with normal breathing interspersed). This frequency is the approximate mid-point of the VLF range and served as a starting point for exploring the effects of rhythmical sighing and the identification of resonance in this frequency range.

A sigh is a deep breath with distinct neurobiological, physiological, and psychological properties. The specific details of what differentiates a sigh from a deep breath or large lung inflation remain uncertain, but it may be defined as an augmented breath that occurs during eupneic breathing and is followed by a respiratory pause called “post-sigh apnea” (Ramirez, 2014). Others define it as a quick deep inspiration with a tidal volume at least twice as large as the mean tidal volume, and a slower expiration (e.g., see Wilhelm, Trabert, & Roth, 2001), with little emphasis on the breath being augmented or followed by apnea. Nonetheless, sighing is a common respiratory phenomenon that can arise spontaneously or be actively produced (e.g., by following instructions). Although sighs are often regarded as a symptom of abnormal or dysregulated breathing (e.g., hyperventilation syndrome (Bass & Gardner, 1985; Berczeller, 1993; Brashear, 1983; Howell, 1990; Lum, 1981; Magarian, Middaugh, & Linz, 1983)), they can prevent reductions in lung compliance and gas exchange caused by breathing with a constant volume (Davis & Moscato, 1994; Ferris & Pollard, 1960; Mcllroy, Butler, & Finley, 1962). Vlemincx, Taelman, Van Diest, and Van den Bergh (2010), Vlemincx, Van Diest, Lehrer, Aubert, and Van den Bergh (2010), Vlemincx, Taelman, De Peuter, Van Diest, and Van den Bergh (2011), Vlemincx, Van Diest, and Van den Bergh (2012), Vlemincx, Van Diest, and Van den Bergh (2012) and Vlemincx et al. (2013) further showed that both spontaneous and instructed sighs restored structured respiratory variability when it was disturbed, such as by stress, emotions, or sustained attention. These latter authors thus defined sighing as a “resetter of the respiratory system”.

We hypothesized that a single sigh would produce immediate, strong oscillations across the cardiovascular system that would fade quickly over time. Further, it was predicted that rhythmical sighing at 0.02 Hz would acutely impose high-amplitude oscillations on multiple cardiovascular parameters in the VLF range, with a spectral peak at 0.02 Hz, that would not be observed in a normal respiration baseline task. The influence of paced sighing on baroreflex sensitivity was exploratory. We also compared the cardiovascular effects of paced sighing to those of 0.1 Hz paced breathing. This paced breathing comparison was included because it is the active element of HRV biofeedback, which has well-characterized physiological and therapeutic effects. These comparisons were exploratory and there were no a priori hypotheses.

2. Method

2.1. Participants

Twenty four young healthy participants (12 women) (mean ± standard deviation: 20.17 ± 1.32 years, range 18–24) who did not drink alcohol were enrolled in the study. Participants were recruited through university and community bulletin boards, electronic postings, and flyers as part of a larger study that aimed at understanding how chronic alcohol use behaviors affect the vasculature. The racial composition was: White (58.3%), Asian (20.8%), Black or African American (12.5%), and mixed or other (8.3%); 16.7% self-reported being Hispanic or Latino. All participants reported being in a good health without respiratory or cardiac disease, or any history of psychiatric disorders; they were not alcohol, nicotine, or recreational drug users. The experiment was approved by the Rutgers University Institutional Review Board for the Protection of Human Subjects Involved in Research. Written informed consent was obtained from all participants and they were compensated for their time spent in the experiment.

2.2. Physiological assessment

A PowerLab Acquisition System (ADInstruments, Colorado Springs, CO) and Finometer MIDI (Finapres, Amsterdam) were used to collect electrocardiogram (ECG), respiration, beat-to-beat blood pressure, and skin conductance. The sampling rate for all data collection was 2000 Hz. A standard lead II was used for ECG measurement. A cuff-sensor for blood pressure measurement was attached to the second phalange of the right middle finger. Skin conductance metal electrodes were attached to the edges of the right palm, one below the thumb and the other below the little finger. Respiratory signals were collected from a single strain belt containing a piezo-electric transducer that was set around the upper part of the chest, just below the underarms. This provided a measure of thoracic breathing.

Respiration was calibrated before the start of physiological records using a standard 800 ml bag. Clips were comfortably set on the nose to ensure that the participants breathed out of their mouths only. They were told to wrap their lips tightly around the edges of the calibration tube to create a closed-loop system with the lungs. They were then asked to completely fill the calibration bag with the air from their lungs and then fully empty the bag. This process was repeated five times. The calibration tube was held in the same position throughout this procedure.

2.3. Procedure

Each participant completed one laboratory session that started between 10 a.m. and 2 p.m. to minimize biological circadian variations. In preparation for the session, participants were asked to get a good night sleep and to eat a light meal prior to, but not within 1 h of the session. Physiological assessment was completed in a sound-attenuated, dimly lit testing room. Participants were seated in a comfortable chair located 1 m in front of a LCD TV screen, and physiological sensors and electrodes were attached. ECG, blood pressure, skin conductance, and respiration were continuously collected while participants completed three tasks. The sequence of tasks in the study included a baseline task, rhythmical 0.02 Hz sighing task, and 0.1 Hz paced breathing task.

The baseline task (B1) was a 5-min low-demand cognitive task (plain ‘Vanilla’ task; Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992) that provides a more standardized baseline than an uncontrolled resting state. A rectangle presented in the center of the TV screen changed color every 10 s for 5-min, and participants were asked to silently count the number of rectangles that were blue in color.

The 0.02 Hz paced sighing task (0.02 Hz-PS) lasted 8.5 min during which participants performed 10 sighs. Participants were instructed to breathe normally until a red screen appeared on the TV, at which point they were asked to start a sigh. The time interval between red screens was every 50 s; the screen remained red for 2 s. The red screen presentation was programmed using E-Prime software (Psychology Software Tools, Inc., Pittsburgh, PA) with an accuracy of ±1 ms. Before starting the recording task, participants were asked to voluntarily perform a few sighs (to determine what was natural for them). They then underwent a short training to become familiarized with the task of sighing at paced intervals. They were informed that during the task, they should sigh naturally. It was emphasized that they should inhale and exhale through their mouth, and to avoid breathing too deeply, which could result in hyperventilation (i.e., feeling dizzy or lightheaded). Respiratory volume of the participants’ sighs was measured from a thoracic respiratory belt and monitored to ensure it was at least twice the volume of breaths taken during the baseline (normal respiration) task. Participants were asked to breathe normally between sighs, remain still other than to sigh along with presentation of the red screen, and avoid speaking during the task.

The 0.1 Hz paced breathing task (0.1 Hz-PBr) lasted 5 min. Participants were instructed to breathe at a rate of approximately six complete breathing cycles per minute by following a visual pacer (Easy Air, Biofeedback Foundation of Europe, Montreal, Canada) presented on the TV screen. The pacer was set to allow for a 5.5 s