



Changes in heart rate variability of flight phobics during a paced breathing task and exposure to fearful stimuli¹

Xavier Bornas² (*Institut Universitari d'Investigacions en Ciències de la Salut IUNICS and Universitat de les Illes Balears, España*),

Jordi Llabrés (*Institut Universitari d'Investigacions en Ciències de la Salut IUNICS and Universitat de les Illes Balears, España*),

Miquel Noguera (*Universitat Politècnica de Catalunya, España*),

Ana María López (*Universidad de Sevilla, España*),

Miquel Tortella-Feliu (*Institut Universitari d'Investigacions en Ciències de la Salut IUNICS and Universitat de les Illes Balears, España*),

Miquel Àngel Fullana (*Universidad Autónoma de Barcelona, España*),

Pedro Montoya (*Institut Universitari d'Investigacions en Ciències de la Salut IUNICS and Universitat de les Illes Balears, España*),

Joan Miquel Gelabert (*Universitat de les Illes Balears, España*), and

Irene Vila (*Universitat de les Illes Balears, España*)

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ABSTRACT. The aim of this experiment was to explore changes in the vagally mediated heart rate variability (HRV) of flight phobics during exposure to feared stimuli. A paced breathing task was included to control for respiration effects. Sixty-one flight phobics (40 women) with a mean age of 39.07 years ($SD = 11.24$) participated in the study. The root mean of the squared successive interbeat intervals differences (RMSSD) was taken as the time domain measure of HRV. High frequency (HF: 0.15-0.4 Hz) and

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² Correspondencia: Departament de Psicologia (UIB). Edificio Guillem Cifre de Colonya. Ctra. Valldemossa Km. 7,5. 07122 Palma Mallorca (España). E-Mail: xavier.bornas@uib.es

low frequency (LF: 0.04-0.15 Hz) band power was calculated on the ECG recordings obtained during free breathing baseline (BL), paced breathing (PB), and exposure (E) to fearful stimuli. Heart rate unexpectedly increased from BL to PB, and decreased from PB to E, while no differences were found between BL and E. No changes in the RMSSD were seen across conditions. HF band power increased, as expected, from BL to PB, and a significant decrease was found from PB to E. LF band power, as well as the LF/HF ratio, increased from BL to E. Discussion focuses on (a) the role of the parasympathetic and the sympathetic nervous systems in fear-related situations, and (b) the effects of paced breathing in preparing the system to cope with threat.

KEYWORDS. Heart rate variability. Flight phobia. Spectral analysis. Paced breathing. Exposure. Experiment.

RESUMEN. En este estudio se analizan los cambios en la variabilidad de la tasa cardíaca (*HRV*) relacionada con el sistema vagal en sujetos fóbicos durante la exposición a estímulos temidos. Se ha introducido una tarea de respiración pautada para controlar los efectos de la misma en la medida de la *HRV*. Han participado en el estudio 61 pacientes con fobia a volar (40 mujeres) con una edad media de 39,07 años ($SD = 11,24$). Como medida de la *HRV* en el dominio del tiempo se ha tomado la *RMSSD* (media cuadrática de las diferencias de los intervalos RR sucesivos). Se ha calculado la potencia espectral en las bandas de alta (HF: 0.15-0.4 Hz) y de baja frecuencia (LF: 0.04-0.15 Hz) sobre los registros de ECG obtenidos durante una línea base sin respiración controlada (*BL*), una fase de respiración pautada (*PB*) y la exposición (*E*) a estímulos temidos. La tasa cardíaca aumentó de forma inesperada de *BL* a *PB*, disminuyó de *PB* a *E*, y no se obtuvieron diferencias entre *BL* y *E*. No se observaron cambios en *RMSSD* entre las tres condiciones. La potencia espectral de HF aumentó, como se esperaba, de *BL* a *PB*, y disminuyó de forma significativa de *PB* a *E*. La potencia espectral de LF, así como la relación LF/HF, aumentó de *BL* a *E*. La discusión se centra en (a) el papel de los sistemas simpático y parasimpático en las situaciones de miedo, y (b) los efectos de la respiración pautada en la preparación del sistema para afrontar estímulos temidos.

PALABRAS CLAVE. Variabilidad de la tasa cardíaca. Fobia a volar. Análisis espectral. Respiración pautada. Exposición. Experimento.

RESUMO. Neste estudo analisam-se as mudanças na variabilidade da taxa cardíaca (*HRV*) relacionada com o sistema vagal em sujeitos fóbicos durante a exposição a estímulos temidos. Introduziu-se uma tarefa de respiração ritmada para controlar os efeitos da mesma na medida de *HRV*. Participaram no estudo 61 pacientes com fobia a aviões (40 mulheres) com uma idade média de 39,07 anos ($SD = 11,24$). Como medida da *HRV* no domínio do tempo tomou-se a *RMSSD* (média quadrática das diferenças dos intervalos RR sucessivos). Calculou-se a potência espectral nas bandas de alta (HF: 0.15-0.4 Hz) e de baixa frequência (LF: 0.04-0.15 Hz) sobre os registos de ECG obtidos durante uma linha de base sem respiração controlada (*BL*), uma fase de respiração ritmada (*PB*) e a exposição (*E*) a estímulos temidos. A taxa cardíaca aumentou de forma inesperada de *BL* a *PB*, diminuiu de *PB* a *E*, e não se obtiveram diferenças entre

BL e *E*. Não se observaram mudanças em *RMSSD* entre as três condições. A potência espectral de HF aumentou, como se esperava, de *BL* a *PB*, e diminuiu de forma significativa de *PB* a *E*. A potência espectral de LF, assim como a relação LF/HF, aumentou de *BL* a *E*. A discussão centra-se em (a) o papel dos sistemas simpático e parassimpático nas situações de medo, e (b) os efeitos da respiração pautada na preparação do sistema para confrontar os estímulos temidos.

PALAVRAS CHAVE. Variabilidade da taxa cardíaca. Fobia a andar de avião. Análise espectral. Respiração ritmada. Exposição. Experiência.

Introduction

In recent years, autonomic inflexibility has been associated with a number of anxiety disorders (Thayer, Friedman, and Borkovec, 1996; Thayer and Lane, 2000; Yeragani *et al.*, 1990), and this has renewed the interest in the psychophysiology of anxiety (Craske, 1999). Some adult anxiety disorders (*e.g.*, panic disorder) have been the focus of much research (Friedman and Thayer, 1998b; Ito *et al.*, 1999; McCraty, Atkinson, Tomasino, and Stuppy, 2001) while others, like specific phobias (*e.g.*, blood phobia, social phobia), have been much less investigated (Friedman and Thayer, 1998a; Grossman, Wilhelm, Kawachi, and Sparrow, 2001). Despite its high prevalence, few studies dealing with flight phobia from a psychophysiological perspective have been published (Capafóns, Averó, Sosa, and López-Curbelo, 1999; Capafóns, Sosa, and Averó, 1997; Capafóns, Sosa, Herrero, and Viña, 1997; Ekeberg, Ellertsen, Seeberg, and Kjeldsen, 1989; Ekeberg, Kjeldsen, Eide, Greenwood, and Enger, 1990; Ekeberg, Kjeldsen, Greenwood, and Enger, 1990; Llabrés, Bornas, Noguera, López, and Barceló, 2005; Wilhelm and Roth, 1998).

Among the psychophysiological markers that have been investigated, vagal tone and vagal reactivity, assessed by measuring heart rate variability (HRV), have been related to a growing list of both physical and psychological disorders in infants, children, and adults -see Beauchaine (2001) for a review-. From a dynamical systems perspective, variability in biological systems is thought to be important because phase transitions often occur at certain critical values when the variability is high. Metaphorically, when the variability of the system is too low (*i.e.*, autonomic inflexibility is high), the system is unable “to shift into an attractor or emotion that is appropriate for a given set of environmental demands” (Thayer and Lane, 2000, p. 203). Decreases in HRV could be taken as indexes of the system’s flexibility loss, and lowered HRV would reflect the system’s lack or deficit of flexibility. Several studies have reported HRV decreases (*e.g.*, Johnsen *et al.*, 2003, in dental phobics during exposure to threatening stimuli) as well as decreased HRV at rest in generalized anxiety disorder (Lyonfields, Borkovec, and Thayer, 1995), blood phobia (Friedman and Thayer, 1998a), and panic disorder (Friedman and Thayer, 1998b).

In spite of these findings, there are still some problematic issues related to the measurement and to the exact meaning of the reported changes in HRV. Heart rate variability can be measured in the time domain with indexes like the average of the

absolute values of successive differences in R-R intervals (MSD) or the root mean of the squared successive interbeat (R-R) intervals differences (RMSSD). Decreases in these measures, however, do not provide information about the underlying autonomic mechanisms that induced such HRV changes. HRV is influenced by both the parasympathetic and the sympathetic branches of the autonomous nervous system (ANS) and therefore it is not possible to know whether HRV is decreased because of parasympathetic withdrawal or due to sympathetic activation, or by a combination of the two branches. Spectral analysis is a helpful aid to get a better knowledge of the role of each branch of the ANS.

HRV can be measured in the frequency domain by calculating the spectral power in different bands. The high frequency band power -HF: 0.15-0.4 Hz, in accordance with the Task Force on Heart Rate Variability, Camm *et al.* (1996)- is exclusively or overwhelmingly mediated by the parasympathetic nervous system (PNS). On the other hand, sympathetic influences predominate in the low frequency band (LF: 0.04-0.15 Hz). Therefore, the study of any changes in these specific bands would contribute toward a better understanding of the autonomic mechanisms underlying anxiety disorders.

Spectral power can be calculated on interbeat intervals time series (*e.g.*, Ito *et al.*, 1999; McCraty *et al.*, 2001; Piccirillo *et al.*, 1997) or on instantaneous heart rate time series (*e.g.*, Goldberger, 1999; Iwanaga, Kobayashi, and Kawasaki, 2005; Yeragani *et al.*, 2002). Both methods should yield similar results (because oscillations in IBIs time series mirror the HR time series waves), although it is difficult to compare them when they are reported in different units (ms^2 or beats^2).

Several studies stress the psychological significance of the sympathetic nervous system (SNS) reactivity. In general, heart rate increases in response to threat are implicitly attributed to the activation of the SNS consistent with a general mobilisation for avoidance or escape behavior. Several measures have been used to evaluate the sympathetic activation. Perhaps the most widely used is the LF band power (*e.g.*, Piccirillo *et al.*, 1997) although it is known to reflect some parasympathetic activity as well. Hayano *et al.* (1991) proposed the LF/HF power ratio as a measure of the sympathetic activity. Sarlo, Palomba, Angrilli, and Stegagno (2002) found marked cardiac activation along with sympathetic increase (using another measure, the T-wave amplitude) when blood phobics were exposed to feared stimuli. Spider phobics, on the contrary, did not show sympathetic activation when confronting a spider-related film. Ito *et al.* (1999) suggest a co-activation of both branches of the ANS in patients with panic disorder in the early stage of the illness.

In HRV studies centered on the role of the PNS in anxiety disorders, controlling for respiration poses another problem. Because the power spectral amplitude of the HF component is influenced by unstable respiration cycles, these cycles should be constantly regulated -as in the study by Ito *et al.* (1999) on panic disorder-. Though full respiratory control is not always possible -*e.g.*, in the study by Iwanaga *et al.* (2005), whose participants had to listen to music, as the respiratory cycle is influenced by musical tempo-, it seems necessary to include a paced breathing period to assess cardiac vagal tone and to control for respiration effects in studies addressed to the evaluation of HF power changes during exposure to threatening situations. The need for respiratory control

when using respiratory sinus arrhythmia (RSA) as an index of vagal tone (HF power is a method to quantify RSA) has been recently emphasized by Tripathi (2004) and by Grossman, Karemaker, and Wieling (1991). In fact, Grossman *et al.* (2001) included a paced breathing phase (at 0.25 Hz) after the baseline phase and before the stressing conditions in their study with older social phobics “to evaluate individual differences in cardiac vagal control” (p. 767). In this study, changes in vagal tone were only found in older female social phobics. Sarlo *et al.* (2002) assessed the basal parasympathetic cardiac control during a 1 min paced breathing task (at 0.13 Hz) before the resting baseline in their study on blood phobia and spider phobia. They found a lowered vagal control in blood phobics as compared to spider phobics. In the study by Ito *et al.* (1999) with panic disorder patients, breathing was paced throughout the entire experimental protocol at 0.25Hz. Changes from supine to tilt position were found in this study, though sympathovagal balance was preserved because of the co-activation of the two branches of the ANS during the tilt test. These studies notwithstanding, respiratory control is not usual in studies on anxiety disorders. As Beauchaine (2001) points out, the use of the term vagal tone in the literature on HRV has been a source of confusion because the validity of measures taken without respiratory control is moderate (p. 188) and they could reflect vagal reactivity instead of vagal tone.

In addition, controlled breathing has been used as a therapeutic strategy in many cognitive-behavioral treatments for anxiety disorders (mainly for panic disorder (PD) but also for specific phobias like flight phobia, *e.g.*, Bornas, Tortella-Feliu, and Llabrés (2006) since “many clinicians believe that symptoms of PD and, to some degree, those of other anxiety disorders are related to respiratory dysregulation” (Wilhelm, Gevirtz, and Roth, 2001, p. 536). These authors also state that “initial evidence indicates efficacy of respiration-focused treatment” (p. 513).

The first aim of this study was to explore changes in the vagally mediated HRV of flight phobics during a paced breathing phase (5 minutes) followed by a period of exposure to feared stimuli (5 minutes). For drawing up this experiment (Montero and León, 2005), we followed the proposal by Ramos-Álvarez and Catena (2004). In a study with dental phobics, Johnsen *et al.* (2003) found HRV (RMSSD) decreases from baseline (uncontrolled breathing) to exposure. In another study with fearful flyers, Bornas *et al.* (2005) also reported RMSSD decreases from baseline (uncontrolled breathing) to some exposure conditions although significant decreases in the HF power were not found. However, as mentioned above, HRV values obtained during a free breathing baseline show great intraindividual variability, making it difficult to assert that further HRV decreases are due to exposure (*i.e.*, they can be related to changes in the respiration rate of the patient instead of stimuli-evoked). In the same way, the absence of significant HRV decreases can be due to the intrinsic variability of the measure during free breathing. Therefore in this study we analyze HRV changes from baseline (uncontrolled breathing) to paced breathing, and from there to exposure.

Secondly, the study aimed to explore changes in the LF component of HRV, as well as in the LF/HF ratio. Assuming that LF power and LF/HF ratio mirror, at least partially, sympathetic activation, the analysis of changes in these measures during the paced breathing and exposure periods could provide significant information on the effects of threatening stimuli on the sympathetic autonomic functioning of flight phobics.

Additionally, we calculated HF and LF band power on both the IBIs and the HR time series in order to determine which of these measures was more sensitive to the experimentally induced HRV changes.

Method

Participants

Sixty-one flight phobics (40 women) with a mean age of 39.07 years ($SD = 11.24$) participated in the study. All of them met the DSM-IV (American Psychiatric Association, 1994) criteria for specific phobia as the main diagnosis and were older than 18. Exclusion criteria were: being in psychological treatment, taking psychotropic medication, suffering from any other psychopathological disorder requiring immediate treatment, having a history of or current psychotic symptoms, suffering from cardiovascular or respiratory illness, or being pregnant. This sample showed high ratings on fear of flying, similar to previous studies on flight phobia. The mean score in the Fear of Flying Questionnaire (see below) was 176.64 ($SD = 35.94$). Bornas *et al.* (2006) reported $M = 179.67$, $SD = 27.00$ for one group and $M = 186.79$, $SD = 26.76$ for another group in a recent study. The sample mean score in the Fear of Flying Scale (see below) was 54.56 ($SD = 12.40$), which is similar to the values reported by Öst, Brandberg, and Alm (1997) for flight phobics completely avoiding flying ($M = 59.0$, $SD = 7.3$) as well as to the values found in Bornas *et al.* (2006): $M = 54.76$, $SD = 8.42$; $M = 57.42$, $SD = 7.10$.

Procedure

Participants were recruited through advertisements in local newspapers. Seventy-two subjects asked for treatment³ and attended the first experimental session at the Clinical Laboratory of the University of the Balearic Islands. Upon arrival, they were given general information about the study and signed a written consent form. Each patient was then seated approximately 1 m from a 17-in. monitor and sensors were attached for psychophysiological recording. A 3-min adaptation phase was followed by a 5-min resting baseline period (BL), a 5-min paced breathing (0.2 Hz) task (PB), and a 5-min period of exposure to a threatening flying sequence (E). The whole experiment was controlled by a computer (Pentium IV 1.6 GHz). Short text messages appeared on the screen before the beginning of each task. During baseline the subject was asked to look at the computer's screen and be relaxed. Five minutes later, the paced breathing task began. The breathing phase was paced by means of a picture of a human face with arrows pointing to the mouth when inspiring and going out from the mouth when expiring; a scrolling bar on the bottom of the screen also indicated the inspiration and expiration phases, which had the same duration. The message "now we need to evaluate your responses to some flying stimuli" came up on the screen before the 5 min. exposure phase. It should be stressed that subjects did not know that feared stimuli would be

³ The present study is part of a full treatment study, so that all participants are getting effective treatment for flight phobia.

presented to them until this moment. A modified version of the take-off sequence of the CAFFT -see Bornas *et al.* (2002) for a description- was used as the threatening stimulus. After exposure, the subject was invited to go to an adjacent room for a clinical interview.

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV); (Brown, DiNardo, and Barlow, 1994) was used to individually assess all subjects. They completed the Fear of Flying Questionnaire (FFQ); (Bornas, Tortella-Feliu, García de la Banda, Fullana, and Llabrés, 1999), and the Fear of Flying Scale (FFS); (Haug *et al.*, 1987). Eleven participants were excluded from the study for clinical reasons (*i.e.*, flight phobia was not the main problem) or physiological reasons (*i.e.*, anomalous ECG signals).

Stimuli and apparatus

The modified take-off sequence of the CAFFT was shown on the screen and sounds were played through speakers. The length of the sequence was 5 minutes. Twenty still pictures with their corresponding sounds (recorded in a real environment) were included. Sessions were conducted in a dimly lit and sound-attenuated room. ECG was recorded placing 10 mm Ag/AgCl electrodes on the left ankle, the right wrist, and the right ankle (Lead II). Instructions were given to subjects in order to minimize arms movements during the experiment. The signal was recorded on a BIOPAC MP150 monitoring system and the sample rate was set to 200 Hz.

Data reduction and analysis

After visual inspection of the ECG recordings to detect anomalous signals, five subjects were excluded from further analysis. An automatic R-wave detector was used to identify the interbeat intervals (IBI) in milliseconds, from which the RMSSD was calculated. The HRV analysis software -version 1.1; Niskanen, Tarvainen, Ranta-Aho, and Karjalainen (2004)- developed by the Biosignal Analysis and Medical Imaging Group at the University of Kuopio was used to calculate the low frequency (0.04-0.15 Hz) and high frequency (0.15-0.4 Hz) power (Fast Fourier Transformation method) on the IBI time series (approximately $N = 300$), as well as the LF/HF ratio.

Instantaneous HR was obtained from the ECG recordings using the algorithm developed by Berger, Akselrod, Gordon, and Cohen (1986) with a sampling rate of 4 Hz, and the low frequency (0.04-0.15 Hz) and high frequency (0.15-0.4 Hz) power were calculated with the Fast Fourier Transformation method on these HR time series ($N = 1100$).

Statistics

A repeated-measures analyses of variance (ANOVA) with one group and three levels (BL, PB, and E) was conducted for each measure. Power measures were ln transformed before any statistical analysis. Alpha level was set to .05 and adjusted (Bonferroni) for multiple comparisons. When sphericity cannot be assumed, *e* values are reported. All the analyses were computed using SPSS 12.0S for Windows.

Results

Means and standard deviations for all the psychophysiological measures for baseline, paced breathing, and exposure conditions are reported in Table 1.

TABLE 1. Means and standard deviations for the psychophysiological measures for baseline, paced breathing, and exposure conditions.

Measures	Condition					
	BL		PB		E	
	M	SD	M	SD	M	SD
HR (bpm)	79.82	11.52	82.71	11.04	80.39	10.82
RR (ms)	766.51	103.56	736.88	99.06	760.91	98.01
RMSSD	25.41	13.19	27.08	14.15	25.49	12.38
ln LF power HR	6.38	.98	6.61	1.03	6.79	.88
ln HF power HR	5.61	.92	6.39	1.00	5.67	.87
ln LF power btb	4.87	1.16	4.93	1.21	5.19	1.07
ln HF power btb	4.28	1.17	4.69	1.13	4.29	1.03
lf/hf ratio	2.44	2.01	2.09	2.84	3.23	2.69

Note. BL: baseline; PB: paced breathing at 0.20 Hz; E: exposure; HR: heart rate (bpm); ln: natural logarithm; HF: high frequency power; LF: low frequency power; RMSSD: root mean of the squared successive R-R differences.

Heart Rate. Each 5 min condition was divided into 30 seconds epochs in order to assess the evolution of HR across the experiment (see Figure 1). During BL the HR values are rather stable. An exponential increase can be observed along the PB phase. HR values decrease gradually during exposure until they reach the baseline level.

The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 12.38$, $p < .001$, $e = .851$. Pair comparisons showed a significant increase from baseline to the PB phase, $t_{(59)} = 4.15$, $p < .001$, and a significant decrease from PB to exposure, $t_{(59)} = 3.54$, $p < .01$.

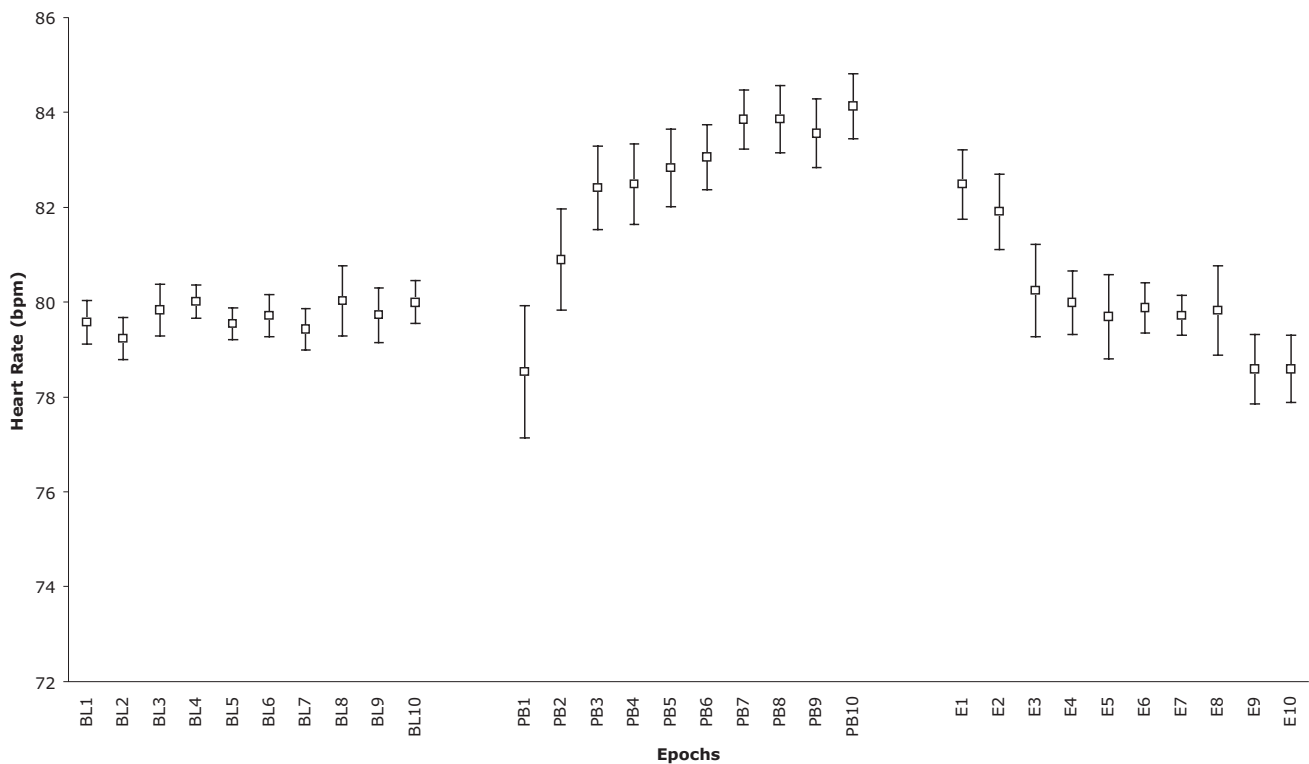
Heart Rate Variability, RMSSD. The repeated-measures ANOVA did not reveal any significant effect.

Heart Rate Variability, spectral analysis:

LF power on HR time series. The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 9.76$, $p < .001$. Pair comparisons showed a borderline increase from baseline to the PB phase, $t_{(59)} = 2.32$, $p = .07$, and a significant increase from BL to exposure, $t_{(59)} = 4.91$, $p < .001$.

HF power on HR time series. The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 60.81$, $p < .001$. Pair comparisons showed a significant

FIGURE 1. Means and standard deviations of HR values over the course of the experiment.



increase from baseline to the PB phase, $t_{(59)} = 9.27$, $p < .001$, as well as a significant decrease from PB to E, $t_{(59)} = 8.93$, $p < .001$.

LF power on IBI time series. The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 4.37$, $p < .05$. Pair comparisons showed a significant increase from baseline to exposure, $t_{(59)} = 3.01$, $p < .05$.

HF power on IBI time series. The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 16.99$, $p < .001$. Pair comparisons showed a significant increase from baseline to the PB phase, $t_{(59)} = 4.92$, $p < .001$, as well as a significant decrease from PB to E, $t_{(59)} = 5.11$, $p < .001$.

LF/HF ratio on IBI time series. The repeated-measures ANOVA revealed a significant condition effect, $F_{(2, 120)} = 6.363$, $p < .01$, $e = .783$. Pair comparisons showed a significant increase from baseline to the exposure phase, $t_{(59)} = 3.02$, $p < .05$, as well as a significant increase from PB to E, $t_{(59)} = 2.83$, $p < .05$.

Discussion

The significant heart rate increase found from baseline to the paced breathing condition was unexpected. To the best of our knowledge, HR increases due to respiratory control at normal rates (*i.e.*, around 0.20 Hz) have not been reported in the literature on anxiety disorders. Respiratory control, however, is not frequent in these studies. Grossman *et al.* (2001), who included a paced breathing phase (at 0.25 Hz) after the baseline phase and before the stressing conditions, do not report any heart rate change during that phase. In the study by Sarlo *et al.* (2002) the paced breathing period occurred

before the free breathing baseline, and they do not inform of any HR change between these periods. Studies with healthy subjects have not found (or do not report) HR increases from free to controlled breathing at normal rates (Badra *et al.*, 2001; Ben Lamine *et al.*, 2004; Sinnreich, Kark, Friedlander, Sapoznikov, and Luria, 1998). Even respiration at very low (0.03 Hz) or very high (0.5 Hz) rates did not affect heart rate in a study with 15 experienced scuba divers by Schipke, Pelzer, and Arnold (1999), who found a stable HR = 72 ($SD = 11$).

Along with the HR increase our results showed an increase in HF power during the paced breathing phase. Hyperventilation could be the cause of such HR increase. However, neither the overt behavior of patients during the paced breathing task nor the spectral analysis lends support to this hypothesis. The respiration rate, as revealed by the HF peak found with the HRV software analysis (Niskanen *et al.*, 2004) during the five min. resting period was 0.23 Hz (*i.e.*, 13.8 breaths/min), which is very close to the 0.2 Hz (12 breaths/min) rate set by the paced breathing procedure. Further, the acceleratory-deceleratory HR cycle was clearly visible within each respiratory cycle on the HR time series during PB. Therefore, subjects were not making deep breaths and they had no trouble to follow the 0.2 Hz rate.

The increase in HR during paced breathing might be due to sympathetic activation. The LF power showed a trend to increase during this period ($p = .07$). However, when power was calculated on the IBIs time series this trend was not so clear. Further, the LF/HF ratio (a measure of sympathetic activation according to Hayano *et al.*, 1991) did not change from baseline to paced breathing.

Cognitive-behavioral models of panic disorder suggest a catastrophic interpretation of body sensations associated to mild anxiety as a mechanism that can lead to more anxiety, hyperventilation, and panic (Wilhelm *et al.*, 2001). In accordance with such models, our subjects could have focused their attention on the respiratory sensations induced by the PB procedure. Then they could have interpreted them as anxiety signs, and this interpretation could lead to the HR increase we observed during the PB condition. There are, however, some problems with this explanation. First, only three subjects in our sample suffered from panic disorder as a co-morbid diagnosis. Catastrophic interpretations can be made, nevertheless, by other anxiety patients (*e.g.*, flight phobics). Second, parasympathetic activation, which can be considered antagonist of the anxiety response, was detected during the PB condition (see below). Subjects were not asked if they felt anxious during PB, but their ANS behavior does not reveal any anxiety. Third, even assuming that some participants focused their attention on their respiratory sensations, the mechanism by which this attentional bias led to the HR increase remains unknown (sympathetic activation was not significant, as we mentioned above). To sum up, we lack a clear explanation of the heart rate increase found during paced breathing. We know that it was not elicited by fear-related stimuli since patients have not been exposed to any threatening situation before nor during this phase. The role of psychological (*e.g.*, attentional) processes and physiological mechanisms has to be elucidated in future studies.

Changes in HR during exposure were also somewhat surprising. It is well known that HR increases during fear episodes (*e.g.*, Sarlo *et al.*, 2002), found HR increases in

both blood phobics and spider phobics when confronting feared films). Our flight phobics, on the contrary, showed a progressive HR decrease during the 5 min. exposure phase. It should be remembered that exposure followed the paced breathing phase, so that HR was very high at the beginning of exposure (82 beats per minute approximately) and a roof effect could prevent further HR increases. Habituation could account for the progressive HR decrease that took place along the exposure time until baseline levels were reached. On the other hand, the effects of exposure on the autonomic system come up when changes in LF power are examined. A significant increase from PB to E was found in LF power (both using HR or IBIs time series for spectral analysis). Further, the LF/HF ratio also increased significantly from BL to exposure. This finding is in agreement with the sympathetic activation found in blood phobics by Sarlo *et al.* (2002) who, nevertheless, did not find the same pattern in spider phobics. Therefore, according to these authors, specific phobias may have specific autonomic reaction patterns, and the flight phobia pattern seems to be more similar to blood phobia than to animal phobia.

Overall, our RMSSD data show a low HRV of the phobic sample as compared to the values reported in other anxiety studies: Johnsen *et al.* (2003) report a RMSSD = 51.00 at baseline and 42.70 during exposure to dental threatening videos; McCraty *et al.* (2001) found 34.32 and 32.13 in panic disorder patients and healthy controls respectively; Bornas *et al.* (2004) reported values of RMSSD = 44.4 and 43.94 in fearful and nonfearful flyers respectively during baseline and 39.17 and 41.79 during exposure to flying images and pictures. Mean values higher than 30 have been published in many other studies for healthy controls as well as for cardiac patients (*e.g.*, Goldberger, 1999; Milicevic, Lakusic, Szirovicza, Cerovec, and Majsec, 2001; Umetani, Singer, McCraty, and Atkinson, 1998). Although the RMSSD values found in the present study are not much lower than the mean values cited by the Task Force (27 ± 12) for healthy subjects, a floor effect could account for the lack of variability decrease during exposure to feared flight situations.

As expected, the high frequency band power showed a significant increase from BL to PB, and a significant decrease from PB to exposure. Such a decrease might be consistent with the HRV decreases found in other studies on specific phobias during exposure phases. However, as we did not find a HF power decrease from baseline to exposure, we cannot know whether the decrease in E was due to emotion or to the removal of the respiratory control after PB. In addition, it is not easy to compare the HF power values reported in different studies because while some of them calculate power on instantaneous heart rate time series (following the widely accepted procedure described by Berger *et al.*, 1986) others calculate the power on time series of interbeat (ms) intervals. In addition, power can be reported in absolute units (beats^2/Hz or ms^2/Hz), relative units (% of power by frequency band), ln transformed, or even normalized units. The values reported by Piccirillo *et al.* (1997) in their study are $\ln LF = 6.4$ and $\ln HF = 4.8$ for the healthy group ($n = 36$), $\ln LF = 5.3$ and $\ln HF = 4.9$ for the less anxious group ($n = 32$), and $\ln LF = 5.2$ and $\ln HF = 4.3$ for the more anxious group ($n = 49$). These values refer to standing position after a tilt test. Bigger *et al.* (1995) reported values for $N = 274$ healthy subjects, aged 40-69, from 24h Holter recordings:

$\ln LF = 6.45$ and $\ln HF = 5.05$. In the study by McCraty *et al.* (2001) $\ln LF = 6.36$ and $\ln HF = 5.03$ are the reported values for healthy controls and $\ln LF = 6.95$ and $\ln HF = 5.19$ for panic disorder patients (daytime). These values are not much different from ours (see Table 1).

As regards the last goal of the study, our results show that calculating power on HR series is more sensitive than calculating power on IBI series. This can be due, however, to the longer length of HR series (1100 points) compared to IBIs time series. The length of these series depends on the heart rate of the subject. Low heart rates (*e.g.*, 60 bpm) will give around 300 values in 5 minutes, while high HR (*e.g.*, 90) would increase the five min series length up to 450 points.

Taken together, the changes observed in HR and in the low and high frequency bands during paced breathing allow for a brief discussion on the role of controlled breathing before confronting threatening situations. As pointed out by Wilhelm *et al.* (2001) controlled breathing has some therapeutic effect (not only in anxiety disorders but also pain disorders). The autonomic mechanisms by which respiration has such effects are far from clear, but based on our results (and in our sample) breathing at 12 during 5 minutes seemingly prepared the organism to better cope with fear. Increased heart rate is a biological adaptive response to confront with threat in open, natural environments. According to the theoretical model of Thayer and Lane (2000) vagally mediated HRV provides the flexibility needed to choose the appropriate behavior in front of a given set of environmental demands. These are the two main effects we observed in flight phobics when respiration rate was controlled at 0.20 Hz. Preparation for fear was not a conscious act since patients did not know that an exposure phase was part of the experimental protocol; it was just induced by the 5 minutes of controlled breathing. In a recent study Bornas *et al.* (2006) compared the efficacy of computer-assisted treatment and a brief nonexposure multicomponent treatment for flight phobia. The multicomponent treatment included breathing training (among other strategies), and the results showed no differences between exposure and nonexposure treatments. Based on the current results the role of breathing training in that study should be reexamined, and it will be the focus of a future study.

To conclude, we speculate that not finding significant increases in HR nor decreases in HF power (vagally mediated HRV) from uncontrolled breathing baseline to exposure can be accounted for by the therapeutic effect of paced breathing. We suggest that PB attenuates the impact of exposure in the patient (system) by preparing it as mentioned above. Exposure in this study simply made HR and vagally mediated HRV values go back to the free breathing baseline levels.

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