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Respiratory control when measuring respiratory sinus arrhythmia during a talking task

Jennifer R. Tininenko*, Jeffrey R. Measelle, Jennifer C. Ablow, Robin High

University of Oregon, United States

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ABSTRACT

The current study explored the effects of talking on respiratory sinus arrhythmia (RSA) during a semi-structured emotional interview (Adult Attachment Interview) using 76 female undergraduates. The effectiveness of 2 different methodological approaches (i.e. talking baseline or transfer function) was explored as respiratory control during talking tasks. RSA was collected during resting baseline, talking baseline, and interview conditions. Subjective reports of distress were higher in the interview than in the other 2 conditions. Mean RSA levels were significantly lower in the 2 talking tasks than in the resting baseline. After applying a transfer function for respiratory control, there were no significant differences between the 3 conditions. Moderator analyses yielded lower RSA values in the talking baseline and interview conditions for participants who reported greater distress during the interview. It was concluded that respiratory controls are likely necessary when using RSA in talking paradigms and that both approaches appeared to be adequate.

1. Introduction

Much attention has been devoted to the study of respiratory sinus arrhythmia (RSA) as a physiological correlate of emotional reactivity and stress response (Allen et al., 2007; Beauchaine, 2001; Berntson et al., 1997; Graziano et al., 2007; Task Force, 1996; Hughes and Stoney, 2000). RSA is generally described as a measure of heart rate (HR) variability in the high frequency (respiratory) band, which reflects time-related cardiac interbeat interval changes that occur within .15–.40 Hz for adults (Berntson et al., 1993). Accordingly, RSA is influenced by complex cardiac and respiratory parameters (Grossman and Taylor, 2007). RSA is derived by quantifying the R–R time series across multiple respiratory cycles. Within normal ranges of respiration, heart rate increases during inhalation and decreases during exhalation as vagal efference to the heart ebbs and flows. However, the amount of HR variability observed across respiratory cycles tends to decrease in times of parasympathetic withdrawal (Berntson et al., 1997). Parasympathetic withdrawal is elicited by a host of external stimuli and during periods of emotional challenge (see Beauchaine, 2001).

The availability of RSA has opened up many interesting avenues for study since it offers a non-invasive, albeit indirect, index of parasympathetic activity during tasks that induce mental or physical stress. Among these, tasks that involve speaking have become favored over non-speaking cognitive challenge paradigms given the ability to assess rapid cognitive processing and interpersonal challenge (Kamarck and Lovallo, 2003) and the similarity to stressors included in individuals' everyday life (Cacioppo et al., 1994), yet rarely have the effects of talking on RSA acquisition been studied. Common speaking tasks include reciting number strings (Uchino et al., 2005), speaking during variations of the Stroop task (Graziano et al., 2007), interview tasks (Obradovic et al., 2010), and the public speaking demands of the Trier Social Stressor paradigm (Altemus et al., 2001; Schubert et al., 2009). Deciding whether or not to control for respiratory variability to prevent the possibility of artificially influencing the measure presents a vexing problem for those interested in studying RSA as a correlate of psychological processes, as was discussed in the 2007 special edition of Biological Psychology (Allen et al., 2007; Denver et al., 2007; Grossman and Taylor, 2007; Porges, 2007b; Ritz, 2009). While some research has suggested that respiratory variability does not sufficiently influence RSA values and therefore does not need to be controlled (Houtveen et al., 2002), other research has shown that as respiratory rate increases and tidal volume decreases, RSA values decrease linearly, independent of cardiac outflow, even while participants are at rest (Grossman et al., 1991; Grossman and Kollai, 1993; Grossman and Taylor, 2007; Ritz, 2009).

Talking in particular may introduce variability into the R–R time series, affecting both the quantification of RSA as well as comparisons of RSA to non-talking baseline periods, both within and across participants. Studies that have measured RSA during

* Corresponding author at: 1200 5th Ave, suite 800, Seattle, WA 98101, United States. Tel.: +1 206 374 0109.
E-mail address: jtininenko@ebtseattle.com (J.R. Tininenko).

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talking tasks have had mixed outcomes. Previously, Cacioppo et al. (1994) found RSA decreases during a talking task while participants were sitting, but found increases in the same task while participants were standing. Conversely, Reilly and Moore (2003) found that RSA increased with talking when they compared RSA values among resting, silent reading, and oral reading conditions. They found that RSA did not differ in the two silent conditions, while it increased during oral reading, a change they attributed to greater velocity and amplitude of inspiratory movement as well as to increased lung volume associated with talking. Both studies suggest that talking may alter inspiration and RSA values sufficiently to warrant experimental controls in studies that include talking. In the present study, we explored further the effects of talking on RSA, and tested two different approaches to respiratory control within the context of a research paradigm that required participants to speak.

2. Respiratory controls

Promising techniques for controlling respiratory effects have been proposed (Grossman and Taylor, 2007; Jennings et al., 1992; Ritz, 2009; Wilhelm et al., 2004). One approach uses paced breathing at pre-established respiratory frequencies, which allows for the acquisition of RSA while controlling for respiration rate, thus leaving only tidal volume uncontrolled (Wilhelm et al., 2004; Ritz et al., 2001). To account for the effects of tidal volume, Wilhelm and colleagues recommend creating an RSA transfer function (RSA_{TF}), which is the ratio of RSA at each respiratory frequency and the tidal volume magnitude associated with each frequency. They argue that these values provide an indicator of individual participants' resting RSA levels devoid of respiratory confounds associated with changes in respiration rate and tidal volume (see Section 3.5 of this manuscript for additional information). A potential strength of this approach is its control of respiratory variability on a breath-by-breath basis rather than covarying mean level respiratory rates with RSA level averaged over an entire task (Berntson et al., 1997; Grossman et al., 1991).

Although this statistical approach is seen by some to be an effective way to manage the effects of respiration on RSA (Berntson et al., 1997; Grossman et al., 1991; Ritz, 2009), others have expressed concern that it may over-correct for respiratory influences, thereby even eliminating meaningful variance in RSA values (Denver et al., 2007; Ritz et al., 2001). Additionally, the paced breathing component of this approach has also been questioned due to the possibility that instructing participants to breathe at specific intervals, in contrast with spontaneous breathing, may induce artificial respiratory conditions and/or emotional processes into the data (Allen et al., 2007; Denver et al., 2007; Porges, 2007a, 2007b).

As proposed in the current study, a talking baseline task that requires periods of prolonged speaking may provide an alternate means of accounting for the effect of respiration on RSA. The purpose of a talking baseline is to provide a neutral talking exercise that can mimic the respiratory pattern of experimentally manipulated talking. Many studies rely on resting baseline periods that are devoid of talking as a comparison to a talking task when computing measures of parasympathetic reactivity. However, this approach to investigating changes in RSA across tasks may largely reflect changes associated with respiratory variability secondary to talking rather than changes directly associated with cardiac variability. As such, a talking baseline may provide an easy and potentially more valid means of assessing RSA change between more comparable situations. In their research of a "vanilla baseline", Jennings et al. (1992) suggested that baselines using a mildly demanding task may be more stable than true resting baselines. A talking baseline may have the additional benefit of mirroring the inhalation pattern of experimental talking tasks without the concerns of inadvertently removing informative experimental variance or introducing artificial variance as in the paced breathing approach. We selected the talking baseline as the closest comparison possible to an interview, an unstructured, spontaneous speech task, rather than using a reading aloud task. Prior research has shown that reading aloud introduces some respiratory and style differences compared with spontaneous speech tasks. This is thought to be an outcome of people using the length of written sentences or passages to anticipate and structure their breaths. Spontaneous speech tasks, such as the talking baseline, do not artificially constrain respiration in this way, and have been associated with increased range of expiratory lung volume, slower rate of speech, and an increased number of filled pauses versus consistent speech in reading tasks (Hodge and Rochet, 1989; Winkworth et al., 1995) Therefore, we anticipated that talking baseline and interview tasks would more closely resemble one another in terms of individual style than a reading aloud task.

The purpose of the present study was to assess two methodological approaches to controlling for respiratory variables and their effects on RSA in talking tasks. The current study compares resting baseline (RB), talking baseline (TB), and an emotional interview (INT) conditions using the control provided by a talking baseline and the transfer function. There were two primary sets of hypotheses in the present study. Although prior research has been inconclusive regarding the direction of RSA change during talking tasks, we had a speculative hypothesis that RSA would be higher during the resting baseline condition than during the talking baseline and emotional interview due to slower respiration rate and greater tidal volume. Furthermore, we predicted that RSA would be higher during the talking baseline condition than during the emotional interview, since participants would experience more distress in the interview condition. This would suggest a successful manipulation, as well as support for the utility of talking baselines when evaluating RSA changes in response to emotional tasks that involve speaking. Second, regarding the transfer function analyses, we predicted that there would be no differences between the resting baseline and talking baseline condition when this respiratory control was applied. However, consistent with the expectation that the interview would elicit a stronger emotional response, we predicted that the transfer function values during the interview condition would be larger than the transfer function values derived in the other more emotionally neutral conditions.

3. Method

3.1. Participants

Seventy-six undergraduate female participants were recruited from an introductory psychology human subjects pool at a university in the US Northwest. Two participants were subsequently excluded from analyses due to equipment malfunctions, thus our final sample size was $N = 74$. The study was limited to female participants since prior studies have found gender differences in cardiovascular measures (Bloor et al., 2004; Grossman et al., 2001). The average age of participants was 20.1 years ($SD = 5.81$), with most being in their first year of undergraduate education. Most participants were Caucasian (85.4%), with Asian (8.3%), Hispanic/Latina (2.1%), and multi-racial (4.2%) participants representing the remainder of the sample. Ten participants spoke English as a second language, but all were sufficiently fluent to complete the study.

Participants' general level of cardiac and respiratory health was assessed on a preliminary questionnaire regarding general health and lifestyle. None of the participants rated themselves in poor health, and 79.2% rated their health as good or excellent. The bulk of the sample participated in at least some form of weekly exercise (.5–3.0 h = 64.6%; 4.0–10.0 h = 33.4%) with only one participant reporting no regular exercise. Participants also rated their typical weekly exertion during exercise as light (33.3%), medium (60.4%), or heavy (6.3%). We requested that participants refrain from engaging in strenuous exercise or activity for an hour prior to arriving at the lab. We did not advise participants to alter their lifestyle behaviors prior to participating in the study, but did collect information on their overall behaviors. Approximately 80% indicated that they did not smoke at all, and only 14.6% indicated that they smoked a "few cigarettes daily". Approximately half of the sample (47.9%) reported not drinking alcohol, and those who did consume alcohol, reported drinking an average of 2.76 (SD = 4.68) drinks weekly. None of the participants endorsed a known heart arrhythmia and 10% endorsed having asthma when asked about history of any other cardiac or respiratory disease.

3.2. Procedure

To ensure consistency and that participants were comfortable, the room was maintained at an average of 22.4 °C. Upon arriving at the lab, each participant gave informed consent to participate, completed a questionnaire on lifestyle behaviors, and was introduced to the physiological acquisition system prior to collecting data. These initial activities were sufficiently long (i.e. at least 15 min) that it is unlikely that effects of any physical activity prior to coming to the lab would be reflected in the data collected. Course credit was provided as compensation for participation. The procedures for each condition are outlined next.

3.2.1. Resting baseline

During the RB condition, participants were asked to sit quietly and watch a 3-min seascape scene on a television monitor. Participants were instructed to focus on the seascape clip, which began after the interviewer left the room. They were also told that the interviewer would return after the clip ended. The interviewer left the room in all study conditions so that her presence would not influence participants. However, participants were continuously monitored by video camera to ensure they were completing each task as instructed.

When the interviewer returned, she instructed participants to complete seven questions on Likert-type scales (1 = not at all; 7 = very much). Participants rated how they felt during the 3-min seascape for 7 emotions (i.e. anxious, happy, depressed, stressed, angry, relaxed, interested). The interviewer was not present while participants completed the subjective measures.

3.2.2. Paced breathing

The second study condition was a 5-min paced-breathing (PB) task in which participants were asked to synchronize their breathing with a series of pre-recorded tones. The waveform was modified to mimic natural breathing (as described in Wilhelm et al., 2004). The purpose of this exercise was to monitor RSA at standardized breathing frequencies within the normal range of breathing. The paced breathing exercise consisted of five continuous speeds of tones (9 (.15 Hz), 12 (.2 Hz), 15 (.25 Hz), 20 (.33 Hz), and 25 (.42 Hz) breaths per minute, BPM), each lasting 1 min. Participants were instructed to continuously inhale as they heard the tone rise, pause when they did not hear a tone, and exhale continuously as the tone fell. To prevent hyperventilation, participants were encouraged to breathe as normally as possible. Each participant practiced for 1 min while breathing with the slowest tone, during which they were corrected if they did not appear to be doing the exercise correctly. All participants were given the opportunity to ask questions before beginning the 5-min exercise.

3.2.3. Talking baseline

The paced breathing exercise was followed by a 3-min TB condition designed to mimic prosody, tidal volume, rate of speech, and respiratory cycles of conversational language. Participants were given a detailed picture book, “The Eleventh Hour: A Curious Mystery” (Base, 1993) and were asked to describe a picture of their choice as if they were “describing it to someone who could not see the page”. Participants were instructed to speak continuously and normally, but not to worry about clarity or content. We elected to have participants produce spontaneous descriptions of the pictures in the book rather than to read aloud so as to minimize the effects of individual differences in how well or comfortable participants were reading aloud. In an effort to further minimize possible discomfort with the task, participants were also assured that many people may feel silly during the exercise, but we were only interested in what happens to them physically while they read.

The interviewer provided a brief example of the exercise by describing one of the illustrations. Participants were told that they were permitted to turn pages so long as they continued their narration (e.g. “I’m all finished with this page, let’s see what’s on the next one”). Participants were instructed to begin talking when the interviewer left the room and to end when the interviewer returned. Immediately after the exercise, participants completed the same subjective rating questionnaire used in the RB condition.

3.2.4. Interview

The first two sections (see below) of the Adult Attachment Interview (AAI; George et al., 1996) served as the emotional condition in which participants were expected to talk continuously, with only very brief interruptions for questions by the interviewer. The AAI is an empirically validated semi-structured interview that assesses mental representations about primary attachment relationships (Hesse, 2008). During the interview, participants are asked to reflect on early attachment experiences and their impact on current functioning. Prior studies have shown that the discussion of attachment during the interview is related to significantly increased physiological activation and emotion from baseline regardless of personal attachment histories (Dozier and Kobak, 1992; Roisman et al., 2004).

The interview began with a participant describing her childhood as far back as she could remember, including details such as where she was raised, family composition, and a general account of her relationship with her parents. Participants were then asked to identify five adjectives that described their early relationship with their mothers and to support each adjective with a specific example of a situation in their early childhood. The exercise was repeated for the participants’ relationships with their fathers. Immediately following the interview, participants were again asked to complete the 7-question subjective rating form.

In an effort to keep comparison conditions as consistent as possible, only 3 min of the INT condition (out of a possible 14–21 min, M = 17.4 min) were used in the analyses. Since interview lengths varied between participants and since physiological measures tend to drift over time, the 3–6 min segment of the interview was used for all participants. This segment was chosen because at this point in the interview, participants were asked to discuss more challenging topics. Furthermore, Kelsey et al. (1999) reported that cardiac reactivity peaks early during prolonged challenge tasks, when there is greater task uncertainty. Choosing the 3-min period early in the interview was intended to capture emotional response during a period of heightened physiological reactivity.[1]

3.3. Data acquisition

For heart period collection, Ag–AgCl disposable electrocardiogram (ECG) electrodes were attached in a tetrapolar fashion on participants’ third rib and the ground electrode to the collarbone. To monitor respiratory activity, a single respiratory air bellows was fit snuggly at the height of the xiphoid process to measure inspiration and exhalation. Participants were asked to breathe into a PVC tube connected to an 800-ml plastic bag to calibrate the respiratory air bellows and to estimate tidal volume during the experimental condition. Participants were given an opportunity to practice breathing into the bag to ensure that they could fill it completely, and then were instructed to repeat the exercise for seven breathing cycles (one inhalation, one exhalation). Skin conductance, pulse and temperature were also measured, however, only RSA, derived from interbeat interval (IBI), tidal volume, and respiration rate were used in the current study.

Physiological responses were collected with a 21-channel Bioamplifier (model JCA-09) manufactured by the James Long Company (Caroga Lake, NY). During the experimental session, ECG and respiration were sampled continuously with low-pass filtering at 1000 Hz. High pass filtering was recorded at .03 Hz. All subsequent processing of IBIs and editing of outliers in the R–R series were carried out using the IBI Analysis System and PHY General Analysis System from James Long (Caroga Lake, NY). Heart rate samples collected every 10 ms were used to calculate mean heart rate per 1-s period. A level detector was triggered at the peak of each R-wave, and the interval between sequential R-waves was calculated to the nearest millisecond. Artifactual epochs were edited manually for each channel for incorrect detection of the R-wave or movement artifacts. For artifact editing, the sampled ECG signal was viewed graphically. When an R-wave was obscured or undetected by the software, a tick mark was manually inserted into the graphical ECG record based on the specific editing rules of Byrne and Porges (1993). Editing the files involved identifying outlier points relative to adjacent data and replacing them by determining the time between successive interbeat intervals. The data were then scanned graphically using the Statistical Analysis System (version 9.1) and outliers were removed. In addition, outliers that were more than 3 standard deviations above or below the mean were removed and replaced with the mean of the episode.

3.4. Data reduction

3.4.1. RSA

RSA was derived using the IBI Analysis System program (the James Long, Co.). In order to obtain heart rate data, interbeat interval was first computed as the interval (in ms) between successive R waves in the ECG. IBI was converted to instantaneous heart rate after editing R–R interval outliers due to movement of artifacts or ectopic myocardial activity. RSA was computed using respiration and IBI data as outlined by Grossman’s peak-valley technique (Grossman, 1983; Grossman et al., 1991). The difference between the minimum IBI during inspiration and the maximum IBI during expiration, in milliseconds, was used to calculate RSA. The difference was computed twice for each respiration cycle, once for inspiration and once for expiration. Using this method, RSA was computed without being impacted by arrhythmia due to baroreceptor, thermoregulation, and tonic shifts in heart rate.

[1] Although prior studies have reported high correlations between the peak-valley and spectral methods, the peak-valley method was selected as the most appropriate means of RSA derivation due to the smaller window of calculation that breath-by-breath analyses affords (Grossman et al., 1990). When calculating the transfer function as a means of respiratory control, the peak-valley method allows for measurable accuracy as it adjusts for respiratory influences on a breath-by-breath basis. If respiration parameters fluctuate during the larger window required by discrete Fourier transformation in spectral analysis, then the mean of the respiration parameters during that window would not yield as accurate a result in the transfer function as a separate measurement of each breath applied to each corresponding RSA measurement.

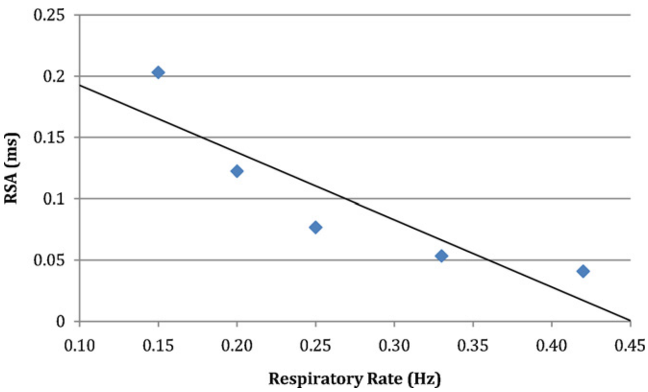


Fig. 1. Mean RSA values during the paced breathing task.

3.4.2. Subjective ratings

Principal components analyses (PCA) were conducted to consolidate participants' subjective ratings following each condition. Separate PCAs of the seven subjective ratings following the TB and the INT conditions yielded identical 2-factor solutions: a distress factor, comprised anxiety, depression, stress, and anger ratings (in both PCAs, eigenvalues > 2.25 and factor loadings > .68), and a positive affect factor, comprised happiness, interest, and relaxation ratings (in both PCAs, eigenvalues > 1.68 and factor loadings > .61). A PCA using participant subjective ratings following the RB yielded the same distress factor but separated the positive affect ratings into two factors, one of which comprised the relaxation ratings alone. Because our interest in the current study was subjective distress during the INT condition, the positive affect ratings were not pursued further. The internal consistency estimates for the continuously measured distress composites were acceptable (Standardized Cronbach alphas/mean inter-item correlations = .69/.42, .75/.43, and .73/.40 for the RB, TB, and INT conditions, respectively).

3.5. Computation of transfer function

The paced breathing condition was analyzed as a means of measuring RSA values at standardized rates of breathing (.15, .20, .25, .33, and .42 Hz). Fig. 1 shows the mean RSA values at the 5 breathing rates. Consistent with prior demonstrations, RSA values declined non-linearly with increasing respiration rates. The results of the paced breathing condition were used to calculate a unique regression line for each participant, which, in turn, provided the basis for the transfer function (TF) calculations (Wilhelm et al. 2004; Ritz and Dahme, 2006).

Prior to calculating TF, RSA data were examined for extreme outliers (most likely attributable to movement artifact) within each respiratory frequency. Locally weighted scatterplot smoothing (Loess) is an approach that weights points on a curve, giving more weight to points directly estimated and nearby points than data points farther away (Cleveland and Devlin, 1988). Loess curve tension factors can be manipulated to change the magnitude of the weight function. To minimize the effect of these outliers on mean TF values, a Loess curve was used with a relatively low tension factor of .075 on the respiratory rate data. The technique was chosen so that all data could be included in the analyses and to improve accuracy of prediction to the regression line.

TF values were then calculated for the 5 paced breathing conditions. The TF calculation was the ratio of RSA to tidal volume (RSA/tidal volume), which reflects the amount of change in RSA amplitude per liter of tidal volume. Under steady-state conditions of the kind presented by our paced breathing task, tidal volume and respiration rate tend to be reciprocally related, so that controlling RSA for one respiratory variable is likely to control for the other (Wilhelm et al., 2004; Ritz and Dahme, 2006). Indeed, we found strong correlations between respiration rate and tidal volume during the five paced breathing rates, absolute values of *r*s > .73, *p*s < .001, at 9 (.15 Hz), 12 (.2 Hz), 15 (.25 Hz), 20 (.33 Hz), and 25 (.42 Hz) breaths per minute.

With TF values calculated for each of the paced breathing rates, intercept and slope parameters were then estimated for each participant by fitting a random-regression equation (using the SAS/STAT Version 9.1 statistical package (Cary, North Carolina) to best represent all five TF means produced by the paced breathing exercise. Generally, the calculated TF regression lines decreased steadily as respiration rate increased. The regression line thus allowed for prediction of transfer function values in the other study conditions. In theory, deviation from this line would indicate RSA changes beyond respiratory effects, presumably, the effects of emotionality or stress.

TF values were then calculated for each instance of RSA in our three experimental conditions (RB, TB, and INT). These TF were also smoothed with a Loess curve with a tension factor of .05 to minimize extreme outliers, while maintaining the bulk (over 92%) of the raw data points in the analyses. We then compared the TF values for each of our three experimental conditions with the TF values predicted by the paced breathing random regression equation. The difference (DIFF) between TF calculated for each condition and the TF predicted from the paced breathing condition ($TF_c - TF_P$) were the comparison values in our central analyses.

3.6. Data analysis and design

In the present study, preliminary and central analyses were largely conducted using repeated measures ANOVAs in which our central within-subjects variable was the condition factor, with either three- (RB, TB, INT) or two-levels (TB, INT) tested. Between-factors and covariates included a native speaker factor (native, non-native) and a continuous measure of emotional distress in response to all three conditions. In these analyses, a significant main effect of condition would indicate RSA differences among our conditions; follow-up Helmert-style contrasts enabled us to locate significant differences. Significant interactions between condition and, for example, participant distress (e.g. Condition × Distress), would signify that RSA values among our conditions differed according to individual differences in levels of distress; posthoc contrasts and *t*-tests enabled us to probe significant interactions. Because sphericity was violated in all analyses, where necessary, *p* values reported below were corrected for nonsphericity using the Greenhouse–Geisser ε.

3.7. Preliminary analyses

Separate repeated measures ANOVAs were tested on RSA, tidal volume, and respiration rate to determine whether there were significant differences in the physiological responses of native and non-native speakers during the three experimental conditions due to potential differences in rate and style of speech, as has been found in prior research (Gut, 2007). We found no significant difference in the responses of native versus non-native speakers in terms of RSA, $F(1.46, 106.68) = .39$, $p = .61$, $\eta_p^2 = .01$, tidal volume, $F(1.34, 97.86) = .46$, $p = .56$, $\eta_p^2 = .01$, and respiration rate, $F(1.11, 81.30) = .12$, $p = .76$, $\eta_p^2 = .00$, respectively.

Participants' rate of speech (words per minute; WPM) in both talking conditions were also evaluated for possible verbal production differences. A word count analysis was completed on 27 (35%) randomly selected participants. Results of a repeated measures ANOVA suggested that there were no differences in WPM between the TB ($M = 174.15$, $SD = 61.95$) and INT conditions ($M = 178.62$, $SD = 62.35$), $F(1, 26) = 2.65$, $p = .12$, $\eta_p^2 = .09$. In a separate repeated measures ANOVA we also tested and found that the interaction between participants' level of distress during the two talking conditions was not significantly related to differences in words per minute, $F(1, 25) = 2.37$, $p > .13$, $\eta_p^2 = .05$. In sum, the lack of overall and distress-related differences in participants' WPM during the talking conditions, coupled with observations that participants had similar speech characteristics in each condition, increased our confidence that the talking baseline was a reasonable comparator to the emotional interview condition.

4. Results

4.1. Preliminary results

We initially evaluated participants' subjective ratings following the three conditions to determine whether the emotional demands of the INT condition were experienced greater than the RB and TB. Although we could not be sure a priori exactly how participants would respond physiologically to the AAI, it was important that we at least confirm that the AAI interview was experienced as stressful as intended. The means and standard deviations for participants' composite rating of distress following each condition are reported in the top portion of Table 1.

A repeated-measures ANOVA revealed a significant effect of condition on participants' subjective ratings of distress, $F(1.22, 101.94) = 14.06$, $p < .001$, $\eta_p^2 = .27$. Follow-up contrasts revealed no differences in levels of subjective distress between the RB and TB conditions, $t(76) = -.83$, $p = .41$, whereas subjective distress during the INT conditions was significantly greater than during the RB, $t(76) = -5.12$, $p < .001$, and TB conditions, $t(76) = -5.03$, $p < .001$, respectively. Importantly, these results indicate that participants responded subjectively to the RB and TB conditions with similarly low levels of distress, yet also show that participants generally responded to the INT condition with significantly greater distress.

4.2. Physiological differences across conditions

The means and standard deviations for RSA, tidal volume, respiration rate levels, and respiratory frequencies during each condition are reported in the middle portion of Table 1. In looking

Table 1
Descriptive statistics for subjective distress, RSA, respiration rate, tidal volume, and transfer function means.

	Condition		
	RB	TB	INT
Subjective distress			
Mean	1.74	1.80	2.37
SD	.68	.76	1.01
RSA (ms)			
Mean	.105	.088	.072
SD	.06	.05	.04
Respiratory rate (s)			
Mean	13.30	13.73	13.90
SD	2.26	2.39	2.60
Tidal volume (L)			
Mean	.62	.59	.42
SD	.45	.32	.25
Respiratory frequency (Hz)			
Mean	.24	.25	.25
SD	.06	.06	.05
Transfer function (ms/L)			
Mean	.08	.12	.16
SD	.52	.52	.51

Note. RB = Resting baseline; TB = Talking baseline; INT = Interview. ms = milliseconds. s = seconds. L = liters.

at ranges of respiratory frequency within our data set, we found that the bulk of the data falls clearly within the high frequency respiratory band, .15 Hz and higher, thereby reflecting the range at which to detect parasympathetic functioning. Less than 10% of data in the resting baseline (Range .12–.40), 2.4% in talking baseline (Range .07–.41), and 5.5% in interview condition (Range .10–.35) fell below .15 Hz. Since so few participants fell into the low frequency band, we feel reasonably confident that we are detecting true parasympathetic effects.

There was a significant effect of condition on RSA, $F(1.46, 108.36) = 10.89$, $p < .001$, $\eta_p^2 = .13$. Helmert-style contrasts showed that RSA during the RB condition was significantly higher than the average of the TB and INT conditions, $F(1, 73) = 13.48$, $p < 001$, $\eta_p^2 = .15$. Contrary to prediction, however, RSA values did not differ significantly between TB and INT, $F(1, 73) = 40$, $p = 05$, $\eta_p^2 = .01$.

Likewise, there was an overall effect of condition on tidal volume, $F(1.34, 99.04) = 6.61$, $p = .006$, $\eta_p^2 = .08$, with Helmert-style contrasts showing a significant difference in tidal volume levels between the RB and the two talking conditions, $F(1, 73) = 7.45$, $p = .008$, $\eta_p^2 = .09$, but a non-significant difference in tidal volume levels between TB and INT, $F(1, 73) = 1.80$, $p = 18$, $\eta_p^2 = .02$. Finally, there was no significant difference in respiration rate among the three conditions, $F(1.12, 82.57) = .57$, $p = .48$, $\eta_p^2 = .00$. In sum, RSA and tidal volume, but not respiration rate, showed possible effects of talking relative to our resting baseline.

Because we had hypothesized sample-level differences in RSA, in particular, we conducted a post hoc two-way (Condition × Distress) repeated measures ANOVA to determine whether RSA levels during the two talking conditions differed based on levels of distress. Furthermore, participants' baseline RSA was covaried to account for possible individual differences in resting state RSA. In separate models, we found that neither distress following the RB nor distress following the TB interacted with the condition factor, $Fs(1, 73) = .41$ and .28, $p > .50$, $\eta_p^2 < .00$, respectively, suggesting that participants had similar levels of RSA during the TB and INT, regardless of their level of distress after the RB or TB. However, reports of distress following the INT condition were related to RSA differences between the two talking conditions, $F(1, 73) = 7.82$, $p = .006$, $\eta_p^2 = .128$. Specifically, as shown in Fig. 2, participants who reported low levels of distress in response to the INT condition had comparable RSA levels during the TB and INT conditions, $t(52) = -.76$, $p = .449$. In contrast, the RSA of participants

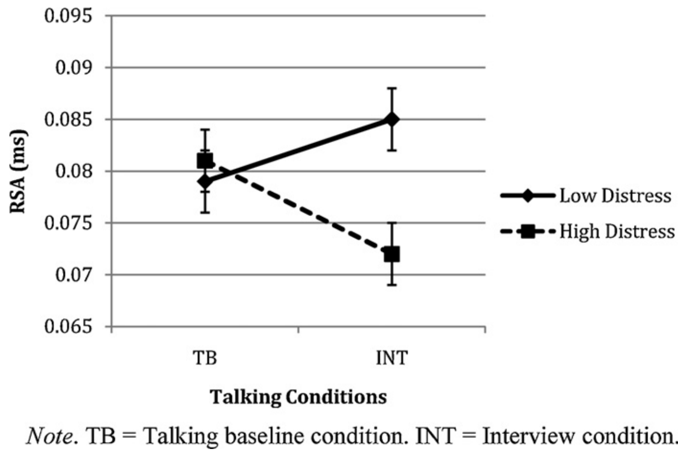


Fig. 2. Interaction between interview distress and RSA means across each talking conditions. *Note*. TB = talking baseline condition. INT = interview condition.

reporting high levels of distress in response to the INT was significantly lower during the INT condition than the TB condition, $t(22) = 2.26$, $p = .044$.

We repeated these analyses on tidal volume and respiration rate to see if distress might moderate the effect of our talking conditions on our other two respiratory parameters. None of the interactions for either tidal volume or respiration rate were significant, all $Fs(1, 73) < .96$, $p > .40$, $\eta_p^2 < .00$. In short, for a subset of individuals who experienced the INT task as distressing, the emotional demands of the AAI produced a significant change in RSA beyond the effects of talking alone, but not in tidal volume or respiration rate.

4.3. Transfer function comparisons

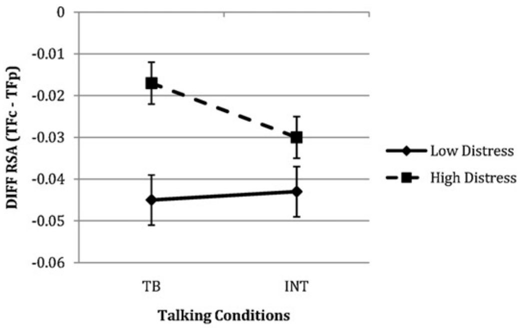
Means and standard deviations of the DIFF values for each condition are presented in the lower portion of Table 1. A repeated-measures ANOVA yielded no significant differences in DIFF values among any of the three conditions, $F(1.39, 107.29) = 1.26$, ns, $\eta_p^2 = .007$ [2]. In other words, after adjusting for the effects of tidal volume variability, there were no differences in the indicators of parasympathetic activity among the three conditions.

As with our mean RSA analyses, we repeated the test of moderation on the DIFF scores. Individual differences in subjects' levels of distress during the RB and TB, $Fs(1, 73) = .84$ and 1.02, $p > .45$, $\eta_p^2 < .003$, respectively, did not moderate change in DIFF values. However, participants' distress in response to the INT did moderate changes in DIFF scores between the TB and INT conditions, $F(1, 73) = 5.29$, $p = .024$, $\eta_p^2 = .078$. As shown in Fig. 3, individuals reporting low levels of distress in response to the INT condition had comparable DIFF scores between the two talking conditions, $t(52) = -.76$, $p = 45$. However, participants in the high distress group showed a marginally significant change in their DIFF scores between the TB and INT conditions, $t(22) = 1.94$, $p = .062$, suggesting that, relative to their transfer function values in the TB conditions, they showed lower than predicted RSA during the INT.

5. Discussion

The central goals of the present study were to contribute to the existing literature base of the influences of talking and respiratory variability on RSA and to investigate the utility of two

[2] A repeated analysis without Loess curve smoothing was computed that yielded an virtually identical F-value.



Note. TB = Talking baseline condition. INT = Interview condition. RSA DIFF (TFc- TFp) = represents the difference (DIFF) between calculated TF and predicted TF from the paced breathing condition (TF_c-TF_P).

Fig. 3. Interaction between interview distress and RSA DIFF scores across talking conditions. *Note.* TB = talking baseline condition. INT = interview condition. RSA DIFF ($TF_c - TF_p$) = represents the difference (DIFF) between calculated TF and predicted TF from the paced breathing condition ($TF_c - TF_P$).

respiratory control procedures, one of which was a novel procedure. Our first objective was to examine respiratory effects during a paced breathing exercise. Our results were consistent with previous findings (Wilhelm et al., 2004), and with the contention that RSA values can be influenced by respiratory parameters (Grossman and Taylor, 2007); as participants increased their rate of breathing, RSA levels decreased systematically. Findings of this sort have led to suggestions that within-individual comparisons of cardiac vagal control across different behavioral tasks might benefit from an adjustment for respiratory confounds (Grossman et al., 1991; Grossman and Taylor, 2007; Ritz, 2009).

Second, we examined mean level differences in RSA across three conditions and found that relative to the resting baseline, both talking conditions produced significant decreases in RSA, suggesting that talking may be an important influence to consider when measuring RSA. Furthermore, this comparison also highlights that a resting baseline may not be a suitable condition in studies that include talking.

Contrary to our hypothesis, however, no significant differences in participants' mean RSA levels emerged between the talking baseline condition and the emotional interview. This unexpected lack of difference in RSA levels between our two talking conditions may be due to potentially more dominant effects of talking, rather than emotional reactivity. However, an alternate, and perhaps more accurate, interpretation of these data would suggest that our interview was not universally experienced as stressful or emotionally arousing. The follow-up moderator analyses showed that, relative to the talking baseline, there was a significant decrease in RSA in response to the interview among participants who rated themselves as more rather than less distressed. Given prior evidence of individual differences in how physiologically and psychologically arousing subjects find the Adult Attachment Interview (Hesse, 2008; Roisman, 2007), we might have been able to detect sample-level differences between our talking and emotional conditions had we used a more universally distressing talking stimulus. These results point to the importance of carefully selected emotion elicitation paradigms in the context of theory driven psychophysiology research (Allen et al., 2007).

A third objective of the study was to compare two methods of respiratory control; a transfer function derived from paced breathing (Grossman et al., 2004; Wilhelm et al., 2004) and a neutral talking baseline. Unlike the analyses with unadjusted RSA values where vagal withdrawal seemed to increase during the talking tasks relative to our resting baseline, there were no longer differences among the conditions in the TF comparisons, suggesting that differences between unadjusted RSA values at baseline and talking conditions may be better attributed to talking induced respiratory variability. These findings are in accord with Grossman and co-workers (Grossman et al., 1991; Grossman and Kollai, 1993) contention that RSA may be influenced by respiratory change thereby artificially introducing variability into the measure.

As with our analyses of unadjusted RSA, the respiratory adjustments associated with the transfer function did not entirely eliminate the emotional effects of the interview. The moderation analyses of the TF values showed significantly greater RSA withdrawal during the emotional interview among the most distressed participants, who presumably experienced the greatest activation and correspondingly would also be the subset experiencing greatest change in RSA. This is an important finding because the transfer function approach has been criticized for potentially eliminating important experimental variability (Denver et al., 2007).

The transfer function may be a useful method since it allows for direct comparison between conditions that may have markedly different rates of respiration. This technique may add flexibility to psychological studies interested in physiological correlates of emotion during talking tasks. Our own and prior evidence (Grossman et al., 2001; Wilhelm et al., 2004) demonstrate that paced breathing procedures are reasonably feasible (i.e. participants can be trained and can perform comfortably) and effective when behavioral demands are limited. It remains to be determined, however, whether respiratory pacing will prove effective when trying to account for respiratory effect in more behaviorally and psychologically demanding conditions.

In sum, the results of the paced breathing and talking baseline procedures were statistically similar, which suggests that either could be a useful means of controlling for possible respiratory influences on RSA during experimental tasks that require speaking. Which to choose? Given that support for the paced breathing and corresponding transfer function approach is elsewhere available (Wilhelm et al., 2004; Ritz et al., 2001), we focus here on making a case for the new talking baseline. First, we found the talking baseline very easy to implement and less complicated to administer than the paced breathing tasks. Although time and experience would likely obviate procedural difficulties, it is a consideration. Second, participants generally experienced the talking baseline as non-distressing, thus it would appear to serve as a face-valid baseline for more emotionally arousing talking tasks. Also, in comparison to the paced breathing task, participants appeared to find the talking baseline less aversive. However, during an early review of this study, it was pointed out that our use of the 20 and 25 breaths per minute frequencies could be experienced as uncomfortable by some, whereas breathing rates of 16 and 18 breaths per minute would generally suffice as the upper limits when estimating a regression line.

Finally, our talking task may offer researchers more than just a baseline condition when conducting research that involves talking. Although our objective was to create a neutral baseline that would yield respiratory rates that (more or less) mirrored our emotional interview, researchers could select different stimuli for the baseline condition depending on the specifics of their experimental task or populations of interest. We hasten to add however, that while interesting, such an adaptation extends beyond the current study, and would need to be the subject of a separate line of inquiry.

5.1. Limitations

The present study is not without limitations. Data were collected on healthy, primarily Caucasian undergraduate females in a university setting, which is a highly specific population and ignores documented differences in males and females cardiovascular activity. Although it is likely that the obtained results are generalizable beyond the immediate study group, we cannot be certain until the study is extended to include males, diverse age groups and ethnicities, as well as more sedentary populations. Our own decision to study females only was a purely practical decision that reflects our laboratory's general focus on women's health and mental health factors.

Another possible limitation is our selection of the Adult Attachment Interview as a stressful talking task given the absence of main effect differences in RSA between the talking conditions. It could be argued that RSA group differences would have emerged in the interview condition had the manipulation effects been stronger. Although the moderator analyses suggested that at higher levels of distress we did see the predicted decline in RSA relative to less distressed participants, it is possible that a more universally stressful talking task might have produced sample levels differences between an emotion eliciting task and a talking baseline of the type tested here. Another possibility, and potential limitation of the study, is our use of self-reports of distress versus interviewer report (e.g. attachment style rating) of distress. Self-report may in fact underestimate the magnitude of the interaction effect found given evidence that some adults tend to minimize distress during the AAI, though show significant physiological arousal (Roisman, 2007). We encourage others to use these approaches with a variety of talking tasks to address the question of magnitude of change beyond what can be attributed to respiration.

One limitation of data acquisition in the current study was that one air bellows was used to collect data on both thoracic and abdominal breathing movements. This method provides an approximate measure of tidal volume, although a two bellows system may provide a more precise measure.

The motivation to assess parasympathetic functioning while participants are engaging in stressful or emotionally activating activities is clear. However, when talking is introduced, it can be difficult to separate effects that are attributable to vagal activity from those due to uncontrolled respiratory differences. The present study points to the need to assess potential influences of respiratory variability on RSA across different conditions that require talking. Although the need to control or not to control for respiratory effect on RSA is complicated and controversial (see Allen et al., 2007), and a precise pronouncement on the subject is beyond the scope of the present study, the current study does offer a promising new talking baseline approach for studies that employ talking tasks.

References

Allen, J., Chambers, A., Towers, D., 2007. The many metrics of cardiac chronotropy: a pragmatic primer and a brief comparison of metrics. Biological Psychology 74, 243–262.

Altemus, M., Redwine, L.S., Leong, Y.-M., Frye, C.A., Porges, S.W., Carter, C., 2001. Responses to laboratory psychosocial stress in postpartum women. Psychosomatic Medicine 63, 814–821.

Base, G. (1993). Eleventh Hour: Harry N. Abrams.

Beauchaine, T.P., 2001. Vagal tone, development, and gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. Development and Psychopathology 13, 183–214.

Berntson, G.G., Bigger, J., Eckberg, D.L., Grossman, P., Kaufmann, P.G., Malik, M., et al., 1997. Heart rate variability: origins, methods, and interpretive caveats. Psychophysiology 34, 623–648.

Berntson, G.G., Cacioppo, J.T., Quigley, K.S., 1993. Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications. Psychophysiology 30, 183–196.

Bloor, L.E., Uchino, B.N., Hicks, A., Smith, T.W., 2004. Social relationships and physiological function: the effects of recalling social relationships on cardiovascular reactivity. Annals of Behavioral Medicine 28, 29–38.

Byrne, E.A., Porges, S.W., 1993. Data-dependent filter characteristics of peak-valley respiratory sinus arrhythmia estimation: A cautionary note. Psychophysiology 30, 397–404.

Cacioppo, J.T., Uchino, B.N., Berntson, G.G., 1994. Individual differences in the autonomic origins of heart rate reactivity: the psychometrics of respiratory sinus arrhythmia and preejection period. Psychophyiology 31, 412–419.

Cleveland, W.S., Devlin, S.J., 1988. Locally weighted regression: an approach to regression analysis by local fitting. Journal of the American Statistical Association 83, 596–610.

Denver, J., Reed, S., Porges, S.W., 2007. Methodological issues in the quantification of respiratory sinus arrhythmia. Biological Psychology 74, 286–294.

Dozier, M., Kobak, R.R., 1992. Psychophysiology in attachment interviews: converging evidence for deactivating strategies. Child Development 63, 1473–1480.

George, C., Kaplan, N., Main, M., 1996. Adult Attachment Interview, 3rd ed. Department of Psychology: University of California, Berkeley.

Graziano, P.A., Keane, S.P., Calkins, S.D., 2007. Cardiac vagal regulation and early peer status. Child Development 78, 264–278.

Grossman, P., 1983. Respiration, stress, and cardiovascular function. Psychophysiology 20, 284–300.

Grossman, P., Karemaker, J., Wieling, W., 1991. Prediction of tonic parasympathetic cardiac control using respiratory sinus arrhythmia: the need for respiratory control. Psychophysiology 28, 201–216.

Grossman, P., Kollai, M., 1993. Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: within- and between-individual relations. Psychophysiology 30, 486–495.

Grossman, P., Taylor, E.W., 2007. Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution, and biobehavioral functions. Biological Psychology 74, 263–285.

Grossman, P., van Beek, J., Wientjes, C., 1990. A comparison of three quantification methods for estimation of respiratory sinus arrhythmia. Psychophysiology 27, 702–714.

Grossman, P., Wilhelm, F.H., Spoerle, M., 2004. Respiratory sinus arrhythmia, cardiac vagal control, and daily activity. American Journal of Physiology Heart and Circulatory Physiology 287, H728–H734.

Grossman, P., Wilhelm, F.H., Kawachi, I., Sparrow, D., 2001. Gender differences in psychophysiological responses to speech stress among older social phobics. Psychosomatic Medicine 63, 765–777.

Gut, U., 2007. Foreign Accent. In: Müller, C. (Ed.), Speaker Classification: Fundamentals, Features, and Methods. Springer, Berlin, pp. 75–87.

Hesse, E., 2008. The Adult Attachment Interview: Protocol, method of analysis, and empirical studies. In: Cassidy, J., Shaver, P.R. (Eds.), Handbook of Attachment: Theory, Research, and Clinical Applications. , 2nd ed. The Guilford Press, New York, pp. 552–598.

Hodge, M.M., Rochet, A.P., 1989. Characteristics of speech breathing in young women. Journal of Speech and Hearing Research 32, 466–480.

Houtveen, J., Rietveld, S., De Geus, E., 2002. Contribution of tonic vagal modulation of heart rate, central respiratory drive, respiratory depth, and respiratory frequency to respiratory sinus arrhythmia during mental stress and physical exercise. Psychophysiology 39, 427–436.

Hughes, J.W., Stoney, C.M., 2000. Depressed mood is related to high-frequency heart rate variability during stressors. Psychosomatic Medicine 62, 796–803.

Jennings, J., Kamarck, T., Stewart, C., Eddy, M., Johnson, P., 1992. Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. Psychophysiology 29, 742–750.

Kamarck, T.W., Lovallo, W.R., 2003. Cardiovascular reactivity to psychological challenge: conceptual and measurement considerations. Psychosomatic Medicine 65, 9–21.

Kelsey, R.M., Blascovich, J., Tomaka, J., Leitten, C.L., Schneider, T.R., Wiens, S., 1999. Cardiovascular reactivity and adaptation to recurrent psychological stress: effects of prior task exposure. Psychophysiology 36, 818–831.

Obradovic, J., Bush, N.R., Stamperdahl, J., Adler, N.E., Boyce, W.T., 2010. Biological sensitivity to context: the interactive effects of stress reactivity and family adversity on socio-emotional behavior and school readiness. Child Development 81, 270–289.

Porges, S.W., 2007a. A phylogenetic journey through the vague and ambiguous xth cranial nerve: a commentary on contemporary heart rate variability research. Biological Psychology 74, 301–307.

Porges, S.W., 2007b. The polyvagal perspective. Biological Psychology 74, 116–143.

Reilly, K.J., Moore, C.A., 2003. Respiratory sinus arrhythmia during speech production. Journal of Speech, Language, and Hearing Research 46, 164–177.

Ritz, T., 2009. Studying non-invasive indices of vagal control: the need for respiratory control and the problem of target specificity. Biological Psychology 80, 158–168.

Ritz, T., Dahme, B., 2006. Implementation and interpretation of heart rate variability measures in psychosomatic medicine: practice against better evidence? Psychosomatic Medicine 68, 617–627.

Ritz, T., Thöns, M., Dahme, B., 2001. Modulation of respiratory sinus arrhythmia by respiration rate and volume: stability across postures and volume variations. Psychophysiology 38, 858–862.

Roisman, G.I., 2007. The psychophysiology of adult attachment relationships: autonomic reactivity in marital and premarital interactions. Developmental Psychology 43, 39–53.

Roisman, G.I., Tsai, J.L., Chiang, K.H., 2004. The emotional integration of childhood experience: physiological, facial expressive, and self-reported emotional response during the Adult Attachment Interview. Developmental Psychology 40, 776–789.

Schubert, C., Lambertz, M., Nelesen, R.A., Bardwell, W., Choi, J.B., Dimsdale, J.E., 2009. Effects of stress on heart rate complexity—a comparison between short-term and chronic stress. Biological Psychology 80, 325–332.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. Circulation 93, 1043–1065.

Uchino, B.N., Holt-Lunstad, Bloor, L.E., Campo, R., 2005. Aging and cardiovascular reactivity to stress: longitudinal evidence for changes in stress reactivity. Psychology and Aging 20, 134–143.

Winkworth, A.L., Davis, P.J., Adams, R.D., Ellis, E., 1995. Breathing patterns during spontaneous speech. Journal of speech and hearing research 38, 124–144.

Wilhelm, F.H, Grossman, P., Coyle, M.A., 2004. Improving estimation of cardiac vagal tone during spontaneous breathing using a paced breathing calibration. Biomedical Sciences Instrumentation 40, 317–324.