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Respiratory Sinus Arrhythmia During Speech Production

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Abstract

The amplitude of the respiratory sinus arrhythmia (RSA) was investigated during a reading aloud task to determine whether alterations in respiratory control during speech production affect the amplitude of RSA. Changes in RSA amplitude associated with speech were evaluated by comparing RSA amplitudes during reading aloud with those obtained during rest breathing. A third condition, silent reading, was included to control for potentially confounding effects of cardiovascular responses to cognitive processes involved in the process of reading. Calibrated respiratory kinematics, electrocardiograms (ECGs), and speech audio signals were recorded from 18 adults (9 men, 9 women) during 5-min trials of each condition. The results indicated that the increases in respiratory duration, lung volume, and inspiratory velocity associated with reading aloud were accompanied by similar increases in the amplitude of RSA. This finding provides support for the premise that sensorimotor pathways mediating metabolic respiration are actively modulated during speech production.

Keywords

respiration; speech motor control; heart rate; reflexes; speech production

Little is known of the sensorimotor pathways mediating respiratory motor control for speech breathing. Although considerable progress has been made identifying areas of the cerebral cortex associated with speech breathing (Murphy et al., 1997), it is unclear how motor commands are conveyed to motoneuron pools controlling respiratory movements. Similarly, little is known regarding the integration of sensory information encoding the state of the respiratory system into the control of speech breathing. The present investigation was undertaken to evaluate the notion that respiratory motor control for speech exploits the low-level sensorimotor mechanisms that mediate metabolic respiration.

The behavioral goals of the respiratory system during speech are distinct from those of rest breathing (Bunn & Mead, 1971; Phillipson, McClean, Sullivan, & Zamel, 1978), resulting in behavior-specific patterns of movement and muscle activity (Hixon, 1973; McFarland & Smith, 1989). Central neural control for behavioral or “voluntary” respiratory tasks, including speech production, involves cortical and forebrain respiratory centers (Euler, 1982; Murphy et al., 1997) whose descending projections occupy separate pathways in the spinal cord from those of the brainstem metabolic pathways (Davis & Plum, 1972). These cortical areas project monosynaptically to spinal motoneurons, where final integration of respiratory inputs takes place (Aminoff & Sears, 1971). These findings have led to the conclusion that brainstem nuclei regulating metabolic breathing patterns are not involved in behavioral or voluntary regulation of respiration (Euler, 1982; Mitchell & Berger, 1975),

which is said to be mediated by corticospinal pathways. With specific regard to speech production, Smith and Denny (1990) found that correlated high frequency oscillations in left and right diaphragm were absent or largely reduced during speech, suggesting a reduction in brainstem respiratory drive.

Electrophysiological recordings from brainstem respiratory neurons, however, suggest that the organization of voluntary respiratory movements involves the integration of brainstem and cortical inputs at the level of the medulla via corticobulbar pathways (Chang, 1992; Orem & Netick, 1986). Marked changes in the firing patterns of respiratory neurons in the medulla have been linked to respiratory changes during vocalization in several species, including guinea pigs (Chang, 1992), cats (Katada, Sugimoto, Utsumi, Nonaka, & Sakamoto, 1996; Zhang, Bandler, & Davis, 1995), squirrel monkeys (Luthe, Hausler, & Jurgens, 2000), and macaques (Larson, Yajima, & Ko, 1994).

In addition to central nervous system effects, metabolic respiratory control relies on a wealth of sensory information to optimize responses to changing homeostatic demands (Euler, 1981). Little is known of the contribution of these low-level sensory inputs to behavioral respiratory tasks such as speech production. The role of bulbar reflexes in the execution of complex movements, including speech, has been discussed in detail without resolution. Abbs and Cole (1982) suggested that orofacial reflexes supporting vegetative functions are incompatible with the motor programs necessary for speech, such that these reflexes must be suppressed during speech production. However, subsequent studies have shown that lip muscle responses to mechanical stimulation can be elicited during speech production and that the amplitude of these responses is not reduced (Smith, Moore, McFarland, & Weber, 1985). No comparable investigation of low-level respiratory reflexes during speech has been reported. Numerous attenuating/facilitating influences on respiratory afferents (Euler, 1981) at least suggest the potential for suppression of low-level respiratory reflex mechanisms. Furthermore, the absence of a Hering-Breuer reflex in adult humans' breathing at normal tidal volumes (Widdicombe, 1961) provides evidence that advanced respiratory control in humans can affect the processing of mechanical feedback from slowly adapting pulmonary stretch receptors (SAPRs).

During metabolic breathing, surface measures of cardiovascular functioning (e.g., heart rate, arterial pressure) exhibit modulation reflecting the interaction of brainstem respiratory and cardiovascular nuclei (Daly, 1986; Richter & Spyer, 1990). Quantification of these cardiorespiratory interactions during speech should provide insight into the activity of brainstem respiratory pathways during speech production.

One prominently observable feature of these interactions is the respiratory sinus arrhythmia (RSA), which consists of an oscillation in the heart rate associated with respiration: Heart rate increases during inspiration and decreases during expiration (Anrep & Rossler, 1936a, 1936b; Hirsch & Bishop, 1981; Saul, Berger, Chen, & Cohen, 1989). The physiological basis of this oscillation is the complex interaction of cardiovascular pathways mediating blood pressure with central and peripheral structures regulating metabolic breathing (Berntson, Cacioppo, & Quigley, 1993; Daly, 1986). The transient increases in arterial blood pressure following each heartbeat elicit reflexive increases in the parasympathetic drive to the heart, thereby decreasing heart rate. These responses are mediated by arterial baroreceptors, specialized sensory nerves that respond to changes in arterial pressure, and medullary cardioinhibitory neurons that control parasympathetic output to the heart. During inspiration, central respiratory inputs in the medulla and peripheral respiratory inputs from SAPRs suppress the firing of the cardioinhibitory motoneurons, consequently increasing heart rate (Davidson, Goldner, & McCloskey, 1976; Gandevia, McCloskey, & Potter, 1978; Gilbert, Jordan, Richter, & Spyer, 1984). During expiration, heart rate decreases as

cardioinhibitory neurons are disinhibited, firing in response to baroreceptor inputs. The result is a rate oscillation characterized by increasing heart rate during inspiration and decreasing heart rate during expiration.

In addition to neural influences, the mechanical effects of respiratory movements contribute to the generation of RSA (Saul et al., 1991; Triedman & Saul, 1994). The alternating increases and decreases in intrathoracic pressure associated with respiration produce similar changes in cardiac output and arterial blood pressure, such that increases in arterial blood pressure resulting from each heartbeat will be greater during expiration than during inspiration. As a result, baroreceptor-mediated decreases in heart rate will be greater during expiration than during inspiration. Again, the result is an inspiratory increase in heart rate followed by an expiratory decrease in heart rate. Lastly, an intracardial atrial stretch reflex makes a small contribution to RSA amplitude that is independent of autonomic innervation (Bernardi et al., 1989; Taha, Simon, Dempsey, Skatrud, & Iber, 1995).

The present study was designed to investigate the role of sensorimotor pathways mediating metabolic respiration in speech breathing. Because these pathways contribute to RSA, quantification of RSA amplitude during speech production and rest breathing should provide insight into their behavior-specific modulation. Specifically, variations in the magnitude and duration of the respiratory cycle can be used to address two theoretical alternatives for observed increases or decreases in RSA amplitude during speech compared to rest. Under homeostatic conditions, increases in lung volume and/or duration of the respiratory cycle produce corresponding increases in RSA amplitude (Hirsch & Bishop, 1981; Saul et al., 1989). Given that participants usually employ greater lung volumes and longer respiratory cycles when reading aloud compared with rest breathing (Bunn & Mead, 1971; Hixon, 1973), the amplitude of RSA would be expected to increase during reading aloud. The absence of these increases during speech would be inconsistent with known established characteristics of RSA (Hirsch & Bishop, 1981; Saul et al., 1989) and would implicate other factors, possibly related to respiratory control, in the gain control of this function during speech. Patwardhan, Vallurupalli, Evans, Bruce, and Knapp (1995) suggested this possibility on the basis of the reductions in RSA amplitude they observed during participants' visuomotor tracking of previously recorded rest breathing traces. On the basis of their findings, one might expect a reduction in RSA magnitude during speech production. Alternatively, an increase in RSA amplitude with increases in lung volumes and/or respiratory durations would suggest modulation of sensorimotor pathways mediating metabolic breathing during speech production.

Method

Participants

Participants included 9 men (age range = 22–37 years; $M = 27.9$, $SD = 5.2$) and 9 women (age range = 24–41 years; $M = 30.1$, $SD = 5.1$). Participants were non-smokers whose medical histories were negative for speech, respiratory, neurological, or cardiovascular disease. To control for potential changes in baroreceptor responsiveness resulting from the ingestion of caffeine (de Mey, Enterling, Brendel, & Meineke, 1987) or a meal (Mosqueda-Garcia, Tseng, Biaggioni, Robertson, & Robertson, 1990), participants refrained from ingesting caffeine for 4 hr and from eating for 3 hr prior to participation in this experiment.

Experimental Protocol

Because this study was primarily concerned with identifying the effects of speech breathing on RSA, it was important to control for other potentially confounding influences on RSA. Increased arousal during tasks such as mental arithmetic can depress baroreflex sensitivity

(Conway, Boon, Jones, & Sleight, 1983) and may alter cardiac responsiveness in ways that are not directly related to respiration. To control for this possibility, an additional condition, silent reading, was included, which involved participants' silent reading of the stimulus material prior to their reading of this material aloud. The order of the tasks was fixed (rest breathing, silent reading, reading aloud) and each was approximately 5 min in duration. All recordings were made in a sound-treated booth. Participants were instructed to read for comprehension and at a comfortable rate during the silent reading condition and at a comfortable rate and loudness level during the reading aloud condition.

Measurements

Electrocardiogram (ECG), rib cage circumference, abdomen circumference, and speech audio signals were recorded for each participant during each of the three conditions. Respiratory movements were measured using respiratory inductance plethysmography (Respirace; Ambulatory Monitoring Inc., Ardsley, NY). The elastic bands of the Respirace ("respibands") were placed around the participant's chest just below the axilla for transduction of rib cage circumference and between the lowest vertebral rib and the iliac crest for transduction of abdominal circumference. The respibands were taped to the participant's skin to prevent slippage during recording. Participants wore loose-fitting pants and a shirt to minimize compression of the abdominal wall by tight-fitting garments. Each participant was seated in a chair adjusted to a comfortable height and was instructed to keep his or her legs in an uncrossed and comfortable position.

Surface electrocardiogram (ECG) was obtained using large surface ($\sim 2.8 \text{ cm}^2$) Ag/AgCl monitoring electrodes (Red Dot, 3M) arranged in standard lead 2 configuration. Speech signals were acquired with an omnidirectional microphone, which was held in an adjustable microphone stand and placed roughly 0.5 m from the participant.

Data Acquisition

ECG signals were amplified (Grass Model 15; gain ranged from 1,000 to 2,000) and band-pass filtered ($f_{bp} = 1\text{--}10000 \text{ Hz}$). Respiratory signals were low-pass filtered ($f_p = 30 \text{ Hz}$) using an analog 4-pole Butterworth filter (Krohn-Hite Model 3364). Audio signals were amplified as necessary using a high quality audio amplifier. Analog outputs from all signals were then filtered for anti-aliasing (RC Electronics Inc., $f_p = 1250 \text{ Hz}$) prior to digitization (2500 samples/s) using a commercially available hardware/ software system (WINDAQ; Dataq Inc., Akron, OH). To remove any computer noise during digitization and to make the data files more manageable, abdomen and rib cage signals were digitally low-pass filtered ($f_p = 22.5 \text{ Hz}$) forward and reverse using a third order Butterworth filter, decimated by a factor of 10, and refiltered.

Signal Processing

Custom routines written for Matlab (Version 5.3; The Mathworks, 1999) were used to calibrate respiratory data, extract target measures, and perform signal averaging. Calibration of the relative contributions of the rib cage and abdomen to instantaneous lung volume used the least squares method (Chadha et al., 1982) after participants performed isovolume maneuvers (Konno & Mead, 1967) at 30%, 50%, and 70% of vital capacity. Lung volume calibration was based on at least three vital capacity maneuvers for each participant. Lung volume changes were recorded as each participant inhaled maximally then exhaled maximally into a spirometer. The calibrated abdominal and rib cage traces of the largest vital capacity maneuver were summed and the difference between the point of maximum and minimum displacement was used to calibrate lung volume to 0% and 100% vital capacity.

For each respiratory cycle, five variables were extracted: inspiratory duration, expiratory duration, respiratory duration (inspiratory duration + expiratory duration), inspiratory lung volume, average inspiratory velocity (inspiratory lung volume / inspiratory duration), and expiratory lung volume. The first derivative of the lung volume signal was used, after low-pass filtering ($f_p = 4.5$ Hz), to determine the onsets and offsets of each respiratory cycle. The onset was operationally defined as the point in the lung volume trace corresponding to the zero-crossing in the velocity signal immediately preceding inspiration; offset was similarly defined as the zero-crossing preceding the following inspiration. The zero-crossing at the peak of the lung volume trace was used to identify end-inspiration.

Temporal marking of heartbeats was accomplished by identification of peaks in the ECG trace that reflect ventricular contraction (i.e., R peaks). ECG signals were first decimated by a factor of 2, yielding an effective sampling rate of 1,250 samples per second and then analyzed using a Matlab routine to identify R peaks in each heart cycle. The time between successive R peaks yielded a time series function (i.e., a tachogram) of corresponding heart period (HP) values. This time series of HPs was interpolated using a linear interpolation function to transform the nonuniformly sampled tachogram into a uniformly sampled array with a new sampling rate of 13.3 samples/s. The interpolated tachogram was registered in time with the lung volume waveform; HP values associated with each respiratory cycle were extracted for subsequent RSA measurement using signal averaging. Respiratory and HP values were extracted from each respiratory cycle that met the following inclusionary criteria: (a) the lung volume trace was free of movement artifact and yawns; (b) the ECG trace was free of cardiac arrhythmias (e.g., premature ventricular contractions); and (c) at least three heartbeats occurred during the respiratory cycle. Mean HP for the 5 min recording sessions associated with each experimental condition was calculated from the original uninterpolated data points. Individual HPs excluded from the RSA analysis were also excluded from the HP analysis.

Spectral analysis of HP time series is the most common technique for estimating RSA amplitude (Malliani, Pagani, Lombardi, & Cerutti, 1991; Saul et al., 1989). Power spectra of short-term (~5 min) resting HP time series typically yield two prominent peaks: a high frequency peak at the respiratory frequency (in the range of 0.2 Hz) that represents RSA and a low frequency peak between 0.04 and 0.12 Hz that is primarily a reflection of cardiac sympathetic drive. Figure 1 (top panel) shows power spectra for a participant's HP data and her lung volume during rest. Two peaks are clearly evident in the HP spectrum; the higher frequency peak coincides with the respiratory frequency. Spectral analysis was unsuitable in the present application because lung volume traces associated with speech production are characterized by broader spectral peaks that are lower in frequency than those associated with rest breathing (bottom panel). As a result, RSA cannot be distinguished from the broad, low frequency energy associated with sympathetic activation (Bernardi et al., 2000). Consequently, signal averaging was used to generate each participant's average HP waveform across the respiratory cycle for each of the three conditions.

Because changes in HP coincide with changes in lung volume only when respiratory frequencies are in the range of 0.15 and 0.25 Hz (Hirsch & Bishop, 1981; Saul et al., 1989), it was necessary to signal average the HP data using three different reference points. The first signal averaging technique (Avg_{insp}) parsed the interpolated tachogram using inspiratory onsets (arrow in Figure 2a), aligning each segment by the HP datum coinciding with end inspiration, which was set to the origin of the coordinate axes (see Figure 2b). When respiratory cycles are shorter (< 4 s), changes in HP lag changes in lung volume and so expiratory increases in HP extend into the next respiratory cycle. For shorter respiratory cycles, then, the expiratory amplitude of the RSA would be underestimated (arrow in Figure 2c). To accommodate this shift in the phase relation between the two data streams, the

second averaging technique (Avg_{exp}) parsed the tachogram using expiratory onsets (see the gray arrow in Figure 2c and 2d). When respiratory cycles are longer (> 10 s), such as during speech, changes in HP lead changes in lung volume (arrow in Figure 2e). As a result, accurate measurement of the inspiratory amplitude of RSA requires parsing the tachogram prior to inspiration (see Figure 2f). To accommodate phase shifts in the two data streams resulting from longer respiratory cycles, parsed segments included the HP values starting 3.3 s after the onset of the preceding expiration (see gray arrow in Figure 2e). This value reflects the minimum duration after expiratory onset that HP decreases would be expected to occur. If the expiratory duration of the preceding respiratory cycle was less than 3.3 s, then no data from the preceding cycle were included.

Three variables were used to quantify RSA at different portions of the respiratory cycle. The inspiratory component of RSA reflects the magnitude of HP decreases associated with inspiration and corresponds to the greater of the two inspiratory amplitudes returned by Avg_{insp} and $Avg_{pre-insp}$. The expiratory component of RSA reflects the magnitude of HP increases associated with expiration and corresponds to the greater of the two expiratory amplitudes returned by Avg_{insp} and Avg_{exp} . The final variable, RSA amplitude, provides a measure of the overall amplitude of the heart rate oscillation across the entire respiratory cycle and was calculated by averaging the inspiratory and expiratory RSA components. Separating RSA into its inspiratory and expiratory components facilitates identification of respiratory variables underlying differences in the magnitude of inspiratory and expiratory changes in HP. In addition, these measured components revealed the maximum change in RSA that might have otherwise been obscured by changes in breathing rates associated with the different experimental conditions.

The average and maximum velocity of inspiratory changes in HP were also calculated. Because the intent of the analysis was to determine the presence of an association between the velocity of inspiratory movements and the velocity of HP changes, the inspiratory component of the Avg_{insp} was used for this analysis. After these values were low-pass filtered ($f_{lp} = 3$ Hz) to remove high frequency directional changes in the HP waveform, the first derivative was calculated. Average and maximum velocity values were then calculated from the data falling between the two zero-crossings.

Statistically significant differences between the three experimental conditions (rest breathing, silent reading, and reading aloud) for all respiratory and HP variables were evaluated using repeated-measures analysis of variance. For several analyses, the assumption of circularity was not met. In these cases, a Greenhouse–Geisser correction was applied to the degrees of freedom of the F statistic to test for main effects between the experimental conditions. When main effects were present, treatment contrasts were used to identify pairwise differences between rest breathing, silent reading, and reading aloud. Cohen's d was also calculated to estimate effect sizes. These values were derived using the original standard deviations instead of the grouped t tests, as recommended by Dunlap, Cortina, Vaslow, and Burke (1996) for repeated-measures designs.

Results

The data set comprised 3,563 respiratory cycles obtained from 18 participants across the three tasks, including 1,168, 1,377, and 1,018 cycles of rest breathing, silent reading, and reading aloud, respectively. Differences in the number of tokens associated with each condition resulted from changes in breathing frequency across tasks. All participants exhibited respiratory modulation of HP consistent with previous studies of RSA; a decrease in HP was observed during inspiration followed by an increase during expiration. These rhythmic oscillations are evident in Figure 3, which shows traces of HP (top panel), lung

volume (middle panel), and the resulting signal-averaged RSA waveform (bottom panel) from Participant 8 during the three tasks.

RSA Amplitude

The depth of modulation of HP across the respiratory cycle was defined as the average amplitude of each participant's inspiratory and expiratory RSA components for each task. Mean RSA amplitudes for all participants were 62 ms ($SD = 57$ ms) during rest breathing, 51 ms ($SD = 37$ ms) during silent reading, and 83 ms ($SD = 45$ ms) during reading aloud. Average lung volumes were calculated by averaging each participant's inspiratory and expiratory lung volumes for each task. Of the 18 participants, 13 increased RSA amplitude during reading aloud when compared with rest breathing, 2 showed no change, and 3 showed a decrease in RSA amplitude. In general, participants who exhibited an increase in RSA amplitude during reading also increased lung volume and respiratory duration, whereas those who exhibited a decrease also decreased one or both of the respiratory variables. Statistically significant differences among tasks were identified, $F(2, 26) = 9.58, p < .005$, and treatment contrasts were used to delineate specific task differences. The top panel of Figure 4 shows the means and 95% confidence intervals accompanying the test for treatment contrasts in RSA amplitude between the experimental conditions. Confidence intervals that do not overlap zero correspond to statistical differences at a criterion p value of .05. RSA amplitude during reading aloud was significantly greater than during rest breathing ($d = .60$), $t(34) = -2.81, p < .01$, and silent reading ($d = -1.15$), $t(34) = 4.32, p < .0001$. No differences were obtained between rest breathing and silent reading ($d = .34$), $t(34) = 1.5, p = .14$.

Significant task effects were also identified for average lung volume, $F(2, 24) = 36.46, p < .0001$. As shown in the middle panel of Figure 4, lung volumes were significantly greater during the reading aloud condition in comparison with rest breathing ($d = .86$), $t(34) = 5.45, p < .0001$, and silent reading ($d = 1.46$), $t(34) = 8.42, p < .0001$. Lung volumes were also greater during rest breathing than silent reading ($d = .68$), $t(34) = 2.97, p < .01$. Significant task effects were also obtained for respiratory duration, $F(2, 34) = 12.81, p < .001$. Respiratory durations were significantly longer during reading aloud than either rest breathing ($d = .58$), $t(34) = 2.03, p = .05$, or silent reading ($d = 1.71$), $t(34) = 5.03, p < .0001$. In addition, respiratory durations were significantly longer during rest breathing than silent reading ($d = .97$), $t(34) = 3.00, p < .01$.

Average HP also exhibited task-related modulation, $F(2, 24) = 38.51, p < .0001$. During reading aloud, HP values were shorter than those of rest breathing ($d = .51$), $t(34) = 6.49, p < .0001$, and silent reading ($d = .62$), $t(34) = 8.36, p < .0001$. No differences in HP between rest breathing and silent reading were found ($d = .13$).

Expiratory and Inspiratory Components of RSA

Expiratory and inspiratory components of RSA and the corresponding respiratory parameters were analyzed separately to determine whether the within-cycle patterns of modulation were consistent with the pattern of modulation across the respiratory cycle. Task-related changes in expiratory lung volumes, $F(2, 23) = 38.08, p < .0001$, and durations, $F(2, 24) = 49.06, p < .0001$, were similar to those observed for lung volume and respiratory duration across the entire respiratory cycle. Participants used significantly greater expiratory lung volumes when reading aloud than during rest breathing ($d = .91$), $t(34) = 5.71, p < .0001$, and silent reading ($d = 1.5$), $t(34) = 8.57, p < .0001$. In addition, expiratory lung volumes were greater during rest breathing than during silent reading ($d = .68$), $t(34) = 2.86, p < .01$. Expiratory durations were significantly greater during reading aloud than during rest breathing ($d = 2.00$), $t(34) = 7.18, p < .0001$, and silent reading ($d = 2.88$), $t(34) = 9.50, p < .0001$. Expiratory durations were also longer during rest breathing than during silent reading

($d = .84$), $t(34) = 2.31$, $p < .05$. Significant main effects were also obtained for the expiratory component of RSA, $F(2, 31) = 40.51$, $p < .0001$. Despite the changes in the volume and duration of expiration, though, no significant differences were identified between the amplitude of the expiratory component of RSA during reading aloud and rest breathing ($d = .41$), $t(34) = 1.96$, $p = .058$. The amplitude of the expiratory component was significantly greater during reading aloud than during silent reading ($d = .99$), $t(34) = 3.66$, $p < .001$. No significant difference was observed between rest breathing and silent reading ($d = .37$), $t(34) = 1.70$, $p = .10$.

The top panel of Figure 5 shows the mean differences and 95% confidence intervals accompanying the test for treatment contrasts in the inspiratory component of RSA between tasks. Also shown are the corresponding differences in inspiratory lung volumes (middle panel), and inspiratory durations (bottom panel). Mean differences and confidence intervals for inspiratory velocities between tasks are shown in Figure 6, top panel. Task-related changes were observed for inspiratory duration, $F(2, 24) = 91.32$, $p < .0001$; inspiratory volume, $F(2, 24) = 34.06$, $p < .0001$; and inspiratory velocity, $F(2, 18) = 116.14$, $p < .0001$. Although respiratory durations were significantly longer during reading aloud than during the other two tasks, inspiratory durations were significantly shorter during reading aloud than during either rest breathing ($d = 3.46$), $t(34) = 13.16$, $p < .0001$, or silent reading ($d = 4.78$), $t(34) = 9.25$, $p < .0001$. In addition, inspiratory duration was greater during rest breathing than during silent reading ($d = 1.00$), $t(34) = 3.91$, $p < .001$. Inspiratory volume was greater for reading aloud than for rest breathing ($d = .80$), $t(34) = 5.11$, $p < .0001$, and silent reading ($d = 1.41$), $t(34) = 8.17$, $p < .0001$. In addition, inspiratory volumes were significantly greater during rest breathing than during silent reading ($d = .67$), $t(34) = 3.06$, $p < .01$. Reading aloud was also associated with greater inspiratory velocities than either rest breathing ($d = 2.96$), $t(34) = 13.22$, $p < .0001$, or silent reading ($d = 2.95$), $t(34) = 13.18$, $p < .0001$. No differences in inspiratory velocity between rest breathing and silent reading were observed ($d = .02$).

These differences across inspiratory measures were accompanied by task-related changes in the amplitude of the inspiratory component of RSA, $F(2, 23) = 11.38$, $p < .005$. Inspiratory components were significantly greater during reading aloud than during rest breathing ($d = .76$), $t(34) = 3.39$, $p < .01$, or silent reading ($d = 1.22$), $t(34) = 4.60$, $p < .001$. No significant difference was observed between rest breathing and silent reading ($d = .31$).

Significant task effects were also observed for both average velocity, $F(2, 26) = 8.69$, $p < .001$, and maximum velocity, $F(2, 19) = 15.83$, $p < .0001$, of inspiratory changes in HP (see Figure 6, middle and bottom panels). The average velocity of inspiratory changes in HP was greater during reading aloud than during rest breathing ($d = .68$), $t(34) = 3.62$, $p < .001$, and silent reading ($d = .64$), $t(34) = 3.60$, $p = .001$. Peak velocities were also significantly greater during reading aloud than rest breathing ($d = 1.06$), $t(34) = 4.55$, $p < .0001$, and silent reading ($d = 1.25$), $t(34) = 5.14$, $p = .0001$. No significant difference in either of these measures was observed between rest breathing and silent reading ($d = .23$). Thus, increases in inspiratory volume and inspiratory velocity during reading aloud were accompanied by similar increases in the amplitude and velocity of inspiratory changes in HP.

Discussion

The results of the present experiment revealed that the increases in lung volume, respiratory duration, and inspiratory velocity usually associated with speech production were accompanied by similar increases in RSA amplitude. These findings are consistent with the well-known effects of tidal volume and respiratory duration on RSA amplitude during rest breathing (Hirsch & Bishop, 1981; Saul et al., 1989) and do not support the idea that output

from respiratory pathways mediating RSA are attenuated during speech. Instead, these results suggest that brainstem nuclei mediating metabolic respiration are involved in the control of speech breathing via sensory feedback from the lungs and/or corticobulbar effects.

At least one other study has investigated the effects of a voluntary respiratory task on RSA amplitude. Patwardhan et al. (1995) used spectral analysis to investigate RSA during 5 min of rest breathing and during a 5-min visual tracking task in which participants tracked their chest wall movements from the rest breathing condition. Results indicated that spectral energy in the region of the respiratory frequency was reduced during the tracking task. The authors suggested that voluntary override of brainstem respiratory pathways may have contributed to the decreases in RSA amplitude. However, these authors suggested that their results could be interpreted alternatively as also supporting the idea that the voluntary control of breathing generates “mental stress” that elicits corresponding increases in heart rate and blood pressure and a reduction in parasympathetic cardiovascular drive. Thus, these investigators suggested that their results might also reflect alterations in baroreflex gain associated with mental stress rather than a reduction in the output of a respiratory central pattern generator.

The increases in RSA amplitude during the reading aloud condition are not consistent with the suggestion that RSA amplitude is reduced during tasks involving behavioral or voluntary control of respiration. Although the control of respiratory movements for speech and for visual tracking may involve overlapping centers of respiratory control, the tasks are considerably different. Visual tracking is a novel task requiring ongoing error correction, which greatly elevates the participant’s awareness of his or her respiratory movements. The respiratory movements that support speech, on the other hand, are overlearned and are relegated largely to automatic control (Netsell, 1982). In addition, prior reading of the control passage silently was intended to minimize the novelty of the reading material during the reading aloud condition. It seems likely that task-specific factors underlie this discrepancy between the present investigation and the work by Patwardhan and colleagues.

Inspiratory and Expiratory Components of RSA

The separate findings for the inspiratory and expiratory components of RSA suggest that the velocity of inspiratory and expiratory movements may also affect RSA amplitude (Strauss-Blasche et al., 2000). The amplitude of the inspiratory component was greatest during reading aloud, which also exhibited the largest inspiratory lung volumes and inspiratory velocities and the shortest inspiratory durations. Although the effects of tidal volume and respiratory duration on RSA amplitude have been extensively investigated, it is only recently that inspiratory velocity has also been implicated in research findings regarding RSA. Strauss-Blasche et al. (2000) had participants manipulate the relative timing of the inspiratory and expiratory phases of their respiratory cycle. These investigators found that short, rapid inspirations followed by long expirations resulted in greater amounts of high-frequency spectral energy and greater mean interbeat decreases in HP than long inspirations followed by short expirations, even though participants actually used greater lung volumes during the latter task. These authors concluded that inspiratory decreases in HP were greater during rapid inspirations than during slower ones. In contrast, the rate of expiration did not appear to affect the magnitude of expiratory changes in HP. The present investigation also measured the effects of increased inspiratory velocity on the velocity of inspiratory HP changes. The increases in both average and maximum velocities of inspiratory HP changes during the reading aloud condition provide further support for the notion that inspiratory velocity influences inspiratory changes in HP.

Parallel increases in the expiratory component of RSA were not observed during the reading aloud condition. During expiration, increases in lung volume and duration associated with

reading aloud were not accompanied by increases in the expiratory component of RSA when compared with rest breathing. Because neural respiratory influences on RSA have been observed only during the inspiration phase of respiration, these findings regarding expiratory RSA do not suggest speech-related suppression of neural output related to metabolic respiratory activity. Other mechanisms related to speech production may have contributed to these findings, however. Van de Borne et al. (2000) found that baroreflex-mediated increases in HP are attenuated during hyperventilation. Bunn and Mead (1971) showed that the physiologic and linguistic demands on the respiratory system during speech cause mild hyperventilation in speakers. Although not measured in the present investigation, hyperventilation by the speakers in this study might account for the failure to identify increases in the expiratory component of RSA between reading aloud and rest breathing.

Respiration and RSA During Rest Breathing and Silent Reading

Despite greater lung volumes and longer respiratory durations during rest breathing than silent reading, no differences were obtained for RSA amplitude between these tasks. This finding contradicts an earlier finding by Bernardi et al. (2000), who found that high frequency (i.e., at the respiratory frequency) spectral energy decreased during silent reading relative to spontaneous breathing. This discrepancy may have resulted from a methodologic difference in measuring RSA. The amplitude of RSA during reading aloud was estimated in the present study using signal averaging, which may have underestimated the differences in HP modulation across tasks compared to spectral analysis.

The finding of decreases in lung volumes and respiratory durations during silent reading was unexpected. Bernardi et al. (2000) also observed decreases in respiratory durations during silent reading, but did not observe differences in lung volume between these two tasks. This discrepancy may reflect further methodologic differences, as Bernardi et al. (2000) derived their volume estimates from uncalibrated respiratory signals that did not account for the differential influences of the abdomen and rib cage on lung volume (Konno & Mead, 1967). These differences nevertheless suggest that during silent reading, participants breathe in a way that is different from rest breathing.

Average HP and RSA During Reading Aloud

The decreases in mean HP observed during reading aloud are consistent with previous reports of increased heart rate during speech (Peters & Hulstijn, 1984; Weber & Smith, 1990). These findings can be accounted for by either an increase in sympathetic output to the heart or a decrease in parasympathetic output to heart. It might seem surprising, then, that RSA amplitude was greatest during the reading aloud condition, given that RSA is almost entirely a result of cardiac parasympathetic modulation. Strauss-Blasche et al. (2000) also observed increases in both RSA and heart rate in their participants during the short inspiration–long expiration condition. These authors suggested that corresponding increases in background levels of cardiac sympathetic activity would account for this finding.

Several investigations have documented speech-related decreases in pulse volume and increases in skin conductance that point directly to an increase in sympathetic output to blood vessels and sweat glands during speech production. If these findings are indicative of a generalized increase in sympathetic drive that includes sympathetic output to the heart, then increases in cardiac sympathetic activity may account for the decreases in mean HP observed in this study during speech. Increases in cardiac sympathetic drive have been associated with reductions in RSA amplitude (Hedman, Tahvanainen, Hartikainen, & Hakumaki, 1995; Taylor, Myers, Halliwill, Seidel, & Eckberg, 2001), suggesting that the RSA amplitudes observed during the reading aloud condition were smaller than those anticipated for respiratory factors alone. Nevertheless, changes in the tonic levels of cardiac

sympathetic and parasympathetic drive are difficult to isolate, such that their contribution to the mean decrease in HP during speech remains speculative.

Implications for Speech Respiratory Control

The observed increases in RSA amplitude during speech provide support for the idea that respiratory drive for speech is mediated by brainstem respiratory nuclei. Support for the possible influences of corticobulbar pathways is derived from changes in the firing patterns of medullary respiratory neurons observed during animal vocalizations (Katada et al., 1996; Zhang et al., 1995). The present results further suggest that the flow of sensory information from the pulmonary bronchi during speech is unmitigated by speech motor programming. Additional support for this suggestion has been provided by Testerman (1970) and Davis, Zhang, and Bandler (1993), who showed in cats that SAPRs mediate reflex responses during vocalizations that are similar to the Hering–Breuer reflexes mediated by these receptors during rest breathing. Vagal feedback was also shown to be important for signaling the transition from inspiration to vocalization (Nakazawa et al., 1997).

Although these studies suggest a role for SAPR feedback in the vocal behaviors of humans, it is highly unlikely to be of the form described by Hering and Breuer in animals, as these reflexes are largely absent in humans (Widdicombe, 1961). During spontaneous speech, Winkworth, Davis, Adams, and Ellis (1995) observed a positive correlation between inspiratory volume and utterance length. During a more controlled speech task, McFarland and Smith (1992) observed that prephonatory movements of the chest wall were directly related to the length of the utterance and to the prevailing lung volume at the initiation of inspiration. In addition, Hixon (1973) reported that participants initiated speech at progressively higher lung volumes with increases in loudness. Inspired lung volume appears to be a controlled variable in speech planning. Given the rapidity of prespeech inspirations, control of inspiratory lung volume seems likely to be influenced by feedback from abdominal and chest wall afferents. SAPRs are likely candidates for this feedback because the discharge properties of these cells reflect transmural pressure (Sant’Ambrogio, 1982), an important variable in establishing target lung volumes for speech.

Study Limitations and Future Directions

Because RSA amplitude is determined by a variety of complex and interacting factors, it is difficult to ascribe task-related changes in RSA to specific factors. For example, it has been shown that the contribution of baroreceptor-mediated changes in heart rate increases from a supine to upright posture (Taylor & Eckberg, 1996), such that the effects of neural factors related to respiration on RSA amplitude could potentially be overshadowed by the contribution of baroreceptor mechanisms to RSA amplitude. It is also unknown whether changes in the relative contributions of the rib cage and abdomen to lung volume during speech (Hixon, 1973) affect the mechanisms for RSA. Additional study of participants in a variety of postures with corresponding measures of blood pressure variability might help to isolate factors such as these. Measurement of RSA amplitude in clinical populations (e.g., double lung transplant patients) might also be useful in revealing specific mechanisms of low-level sensorimotor modulation during speech.

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Figure 1.

Top: power spectra of short-term (~5 min) resting heart period (HP; gray) and lung volume (black) time series. The lung volume power spectrum exhibits a well-defined peak at the breathing frequency that corresponds to the high frequency peak (i.e., respiratory sinus arrhythmia) in the HP spectrum. In addition, a lower frequency peak is evident and reflects cardiac sympathetic activity. Bottom: corresponding power spectra for HP and lung volume time series during speech production. The spectral peak for the lung volume data is broader and lower in frequency during speech, rendering it indistinguishable from the lower frequency energy in the HP spectrum associated with sympathetic activity.

Figure 2.

The three signal averaging methods used to calculate respiratory sinus arrhythmia (RSA). Heart period plots display both the raw time series (—) and the interpolated time series (●). When changes in respiration are approximately 180° out of phase with changes in heart period (a), it is sufficient to parse the heart period data at the onset of inspiration (black lines) and average these traces (b). The origin of the axes corresponds to the point of end inspiration in the respiratory cycle. At higher breathing rates (c), changes in respiration lag changes in heart period (black lines), requiring that the heart period data be parsed at end inspiration (gray lines) and averaged (d). Again, the origin corresponds to end inspiration. At slower breathing rates (e), changes in respiration lead changes in heart period (black lines), requiring that the heart period data be parsed 3.3 s after the onset of the previous expiration (gray lines) and averaged (f). The origin of the axes corresponds to the onset of inspiration.

Figure 3. Raw (—) and interpolated (●) heart period time series (top panels), respiratory traces (middle panels), and RSA (bottom panels) from one participant for all three experimental conditions. The signal averaged RSA waveforms displayed for each task were calculated using Avg_{insp} . For the reading aloud trace, a portion of the $Avg_{pre-insp}$ waveform was appended to the beginning of the trace as this represented the larger of the two inspiratory amplitudes.

Figure 4. Mean differences and 95% confidence intervals between tasks for RSA amplitude (top), lung volume (middle), and respiratory duration (bottom) across experimental conditions.

Figure 5. Mean differences and 95% confidence intervals between tasks for the inspiratory component of RSA (inspiratory RSA; top), inspiratory lung volume (middle), and inspiratory duration (bottom) between experimental conditions.

Figure 6. Mean differences and 95% confidence intervals between tasks for inspiratory velocities (top) and average (middle) and peak (bottom) velocity of inspiratory changes in heart period (inspiratory RSA) across experimental conditions.