Influence of Continuous Speaking on Ventilation

Jeannette D. Hoit
Heather L. Lohmeier
National Center for Neurogenic Communication Disorders
and
Department of Speech and Hearing Sciences
The University of Arizona
Tucson

This study was conducted to explore the influence of speaking on ventilation. Twenty healthy young men were studied during periods of quiet breathing and prolonged speaking using noninvasive methods to measure chest wall surface motions and expired gas composition. Results indicated that all subjects ventilated more during speaking than during quiet breathing, usually by augmenting both tidal volume and breathing frequency. Ventilation did not change across repeated speaking trials. Quiet breathing was altered from its usual behavior following speaking, often for several minutes. Speaking-related increases in ventilation were found to be strongly correlated with lung volume expenditures per syllable. These findings have clinical implications for the respiratory care practitioner and the speech-language pathologist.

KEY WORDS: hyperventilation, end-tidal P_{CO_2}, breathing, respiratory kinematics, magnetometers

During speaking, the breathing apparatus generates pressure to drive the larynx and upper airway structures to create the sounds of speech, while at the same time serving to meet gas exchange requirements. It is well established that the behavioral strategies used to accomplish this dual task differ substantially from those used for quiet breathing. When compared to quiet breathing, breathing for speech is characterized by shorter inspirations and longer expirations, larger lung volume excursions, and higher and more sustained (expiratory) alveolar pressures (Bouhuys, Proctor, & Mead, 1966; Hixon, Goldman, & Mead, 1973; Hixon, Mead, & Goldman, 1976). These characteristic behaviors are designed to meet the demands of spoken communication. For example, short inspirations and long expirations help to minimize the time spent in silence (inspiring) and extend the time spent speaking (expiring) so that the speaker can better "hold the floor" during conversation. As another example, the regulation of alveolar pressure helps to maintain speech loudness at a relatively constant level.

These speaking-related breathing behaviors influence ventilation. The majority of studies to date have shown that speaking causes ventilation to increase above the level of ventilation associated with quiet breathing (Abel, Mottau, Klubendorf, & Koepchen, 1987; Bunn & Mead, 1971; Meanock & Nicholls, 1982; Warner, Waggener, & Kronauer, 1983). This observation has been interpreted to indicate that speaking causes "hyperventilation." This interpretation has been based on measures of ventilation (i.e., the amount of gas moving into or out of the lungs, sometimes called total ventilation) (Abel et al., 1987; Meanock & Nicholls, 1982), estimates of alveolar ventilation (i.e., the amount of gas that participates in the exchange of oxygen and carbon dioxide, calculated by

subtracting estimated dead space ventilation from total ventilation) (Warner et al., 1983), and measures of total ventilation and estimates of alveolar ventilation combined with measures of the partial pressure of carbon dioxide (P_{CO_2}) in expired air (Bunn & Mead, 1971). In contrast to previous work, one recent study showed speaking (at comfortable loudness) to be associated with "hypoventilation," based on measures of expired P_{CO_2} (Russell, Cerny, & Stathopoulos, 1998).

The present investigation was conducted to examine the influence of speaking on ventilation in a way that might offer insights into the discrepancies among previous reports. It also was designed to extend previous work by (a) elucidating the range of individual subject behavior across a large group of homogeneous subjects, (b) determining if ventilation changes across repeated speaking performances, and (c) determining if the act of speaking alters subsequent quiet breathing patterns.

Method

Subjects

Twenty healthy men, age 22 to 27 years, served as subjects. Selected physical characteristics of the subjects are given in Table 1. They were first-language American English speakers with normal speech, language, and hearing. All subjects passed an audiometric screening test for 0.5, 1.0, 2.0, 4.0, and 8.0 kHz presented at 25 dB SPL.

Table 1. Selected physical characteristics of the subjects.

Subject	Height (cm)	Weight (kg)	Vital capacity (L)	Resting expiratory level (%VC)
1	198.1	79.8	4.9	35.7
2	200.7	88.5	6.0	41.2
3	181.6	78.9	4.8	37.9
4	185.4	84.8	6.3	27.0
5	170.2	69.2	4.6	29.7
6	175.3	66.7	5.2	39.4
7	170.2	65.3	4.7	31.9
8	181.0	87.1	5.9	30.8
9	182.9	74.8	5.3	34.0
10	180.3	77.1	5.0	36.0
11	186.7	73.5	5.7	47.4
12	185.4	81.2	5.6	40.2
13	189.9	62.6	4.3	41.9
14	176.5	80.5	5.1	30.7
15	191.8	102.3	6.5	27.1
16	182.9	72.6	5.9	42.4
17	182.9	79.4	4.1	32.9
18	177.8	95.9	4.1	20.7
19	175.3	59.4	4.0	38.0
20	170.2	64.0	4.6	39.1

All subjects were in good respiratory health as determined by responses to a questionnaire and performances on selected respiratory function tests. Specifically, they were free of signs and symptoms suggestive of respiratory disease, without skeletal disease or abnormality affecting the chest wall, without history of surgery or injury involving the breathing apparatus, without history of smoking (except for Subject 3 who had quit several years before the study), without history of prolonged exposure to high levels of secondary smoke or toxic agents, and free of allergies or respiratory infections on the day of testing. Respiratory function tests consisted of forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1). Subjects were required to generate an FVC and an FEV_1 that were at least 80% of the predicted value (Miller, 1986) for healthy men of corresponding age and height (Knudson, Lebowitz, Holberg, & Burrows, 1983).

To ensure that expired CO_2 could be sensed easily at the anterior nares, nasal pathway resistance was estimated for each subject on the day of testing. This was done by having the subject inspire deeply and expire passively through the nose while oral pressure and nasal flow were recorded. Nasal pathway resistance was calculated at a flow of 0.25 L/s and was less than 5 cm H_2O/L/s for all subjects.

Data Recordings

Recordings were made of surface motions of the chest wall, estimated blood-gas levels, and the acoustic speech signal. Surface motions of the chest wall were sensed with respiratory magnetometers (GMG Scientific, Inc., 1980) using the method of Hixon et al. (1973). These magnetometers included two generator-sensor coil pairs, one for the rib cage and one for the abdomen. The generator coil of each pair was attached to the front of the torso at the midline, one at sternal midlength, and one just rostral to the umbilicus. The sensor coil in each pair was attached to the back of the torso at the midline at the same axial level as its generator mate. Output signals from the two sensors were processed electronically and monitored online with a storage oscilloscope.

Partial pressure of carbon dioxide (P_{CO_2}) and oxygen saturation (SpO_2) were monitored continuously (Ohmeda 4700 Oxicap Monitor, Madison, WI). P_{CO_2} was sensed via a double-barreled cannula placed just inside the subject's anterior nares and connected on the other end to an infrared analyzer. P_{CO_2} was displayed as both a waveform and a numeric average of end-tidal P_{CO_2} (mmHg). SpO_2 was sensed with a finger oximeter probe and was displayed as a numeric percentage. Calibration of P_{CO_2} was conducted according to manufacturer's instructions

immediately before each data recording session.

The acoustic speech signal was sensed by an air microphone (AKG C451EB, AKG Acoustics, Wein, Austria) and recorded along with the magnetometer (rib cage and abdomen) and P_{CO_2} signals on a videocassette data recorder (Vetter 820, Rebersburg, PA). Video signals also were recorded on another channel of the data recorder. SpO_2 percentages were recorded by hand.

Performance Tasks

The subject was seated upright in a chair especially designed to accommodate the dorsal magnetometer coils. Each subject performed chest wall maneuvers, quiet breathing, and speaking.

Chest Wall Maneuvers

Chest wall maneuvers included isovolume, vital capacity, and relaxation maneuvers. Isovolume maneuvers were performed to determine the functional relationships between the motions of the rib cage and abdomen from which volume-motion relationships were determined. They were executed by having the subject close the larynx at the resting expiratory level (REL) and displace volume between the abdomen and rib cage by contracting and relaxing the abdominal muscles. Isovolume maneuvers were performed at the beginning and end of the recording session.

Vital capacity maneuvers were performed by having the subject, wearing a noseclip and coupled to a spirometer (Warren E. Collins 9-liter spirometer, Braintree, MA), inspire fully from REL and then expire fully. The largest volume expired over three trials was taken as the subject's vital capacity. This procedure also provided measures of the subject's inspiratory capacity and expiratory reserve volume. Additional spirometric calibrations of the magnetometer signals were made near the subject's REL (e.g., 1 L above REL and 0.5 L below REL) to ensure accuracy of volume measures within the range where quiet breathing and speech breathing were expected to occur.

Relaxation maneuvers were included to help ensure that the subject had not shifted posture. For these maneuvers, the subject was instructed to stop breathing at REL and to completely relax the breathing muscles.

Quiet Breathing and Speaking

Following the chest wall maneuvers, the subject performed a series of alternating quiet breathing and speaking trials. The subject was instructed to maintain a constant posture when performing these tasks. Occasional breaks were provided during which the subject was allowed to move.

For all quiet breathing trials, the subject was instructed to sit quietly with eyes open and mouth closed. The first trial consisted of 5 min of continuous quiet breathing. Hereafter, this first trial of quiet breathing is referred to as "baseline" quiet breathing. The remaining three trials immediately followed periods of speaking and lasted at least 5 min and not more than 10 min. These are called "post-speaking" quiet breathing trials. Each of these three trials was terminated after the subject's end-tidal P_{CO_2} was judged to have returned to baseline values.

For the speaking trials, the subject was instructed to read continuously in his usual voice until told to stop. The reading passage was a modified version of a story about meteors and meteorites written at a fifth-grade level (Lauber, 1989). This passage was chosen because it presented a simple reading task for literate adults and because it was low in emotional content, thereby avoiding the influence of emotional arousal on speech breathing behavior (Goldman-Eisler, 1955; Reynolds & Paivio, 1968). As soon as 10 min had elapsed, the subject was told to stop speaking and to sit quietly.

Data Analysis

For baseline quiet breathing, data from the final 4 min of the 5-min recording period were analyzed. The initial minute was eliminated from the analysis because breathing was typically less regular than during subsequent minutes and the interest was in obtaining a sample of stable quiet breathing for the baseline condition. For the three speaking trials and the post-speaking quiet breathing trials, all data were analyzed. All measurements (except SpO_2) were made on a breath-by-breath basis using a computer (Macintosh Quadra 950, Apple Computer, Inc., Cupertino, CA) and customized software (LabVIEW, National Instruments Corporation, Austin, TX).

Chest wall kinematic signals were calibrated and converted to volumes using procedures described in detail elsewhere (e.g., Hixon et al., 1973; Hoit & Hixon, 1987). Rib cage and abdomen signals were examined qualitatively using motion-motion graphs. Measurements were made from time-motion plots of calibrated lung volume signals (summed rib cage and abdomen signals) displayed along with the acoustic speech signal. Tidal volume (L) was determined by measuring the lung volume excursion for the inspiratory phase of the breathing cycle. Breathing frequency (breaths/min) was calculated by dividing 60 by the duration of the entire breathing cycle, and minute volume (L/min) was calculated from the product of inspiratory lung volume excursion and breathing frequency. The number of syllables produced per breath group (sylls/breath group) was counted. Average speaking rate (sylls/s) was calculated

by dividing the number of syllables by expiratory time (speaking continued throughout expiration). Average lung volume expended per syllable (ml/syll) was calculated by dividing the number of syllables produced by the expiratory lung volume excursion.

Measures of end-tidal P_{CO_2} (mmHg) were obtained from the partial pressure of CO_2 during the alveolar plateau. To meet the criterion for an acceptable plateau, the partial pressure of CO_2 had to remain stable for at least 0.4 s. End-tidal P_{CO_2} measures were obtained for quiet breathing only. No measures were made during speaking because acceptable plateaus were rarely generated. Instead, speaking-related measures of end-tidal P_{CO_2} were obtained during the first 30 s of quiet breathing that immediately followed cessation of speaking (representing 2 to 6 breaths).

Results

SpO_2 was 95% or higher for all subjects throughout data collection sessions and did not differ between quiet breathing and speaking. Measures of minute volume, tidal volume, breathing frequency, and end-tidal P_{CO_2} were highly similar across the three speaking trials, as shown in Table 2. Analysis of variance indicated no significant differences across speaking trials for these ventilation-related measures [minute volume: $F(19, 2) = 0.41$, $p = 0.67$; tidal volume: $F(19, 2) = 2.26$, $p = 0.12$; breathing frequency: $F(19, 2) = 1.09$, $p = 0.35$; end-tidal P_{CO_2}: $F(19, 2) = 0.12$, $p = 0.73$]. Therefore, measures from all three speaking trials were combined for subsequent analyses.

Individual subject means and standard deviations for ventilation-related measures during quiet breathing and speaking are displayed in Figure 1. Regarding minute volume, the group mean was 7.15 L/min (range of individual subject means: 4.54 to 10.33 L/min) for quiet breathing. Speaking involved larger minute volumes, with a group mean of 13.94 L/min (range: 8.77 to 21.91 L/min), representing a group mean increase of 6.79 L/min (range: 1.36 to 15.21 L/min) above quiet breathing levels. For the majority of subjects, the increase in minute volume during speaking was accomplished by increasing both tidal volume and breathing frequency. For the remaining subjects, the minute volume increase for speaking was accomplished by augmenting either tidal volume (Subject 11) or breathing frequency (Subjects 5, 7, 8, 10, 14, 16, 17, and 20). Stated differently, 12 subjects increased tidal volume and 19 subjects increased breathing frequency from quiet breathing to speaking. For quiet breathing, tidal volume averaged 0.65 L (range: 0.34 to 1.16 L, representing 6.94% to 25.22 %VC) and breathing frequency averaged 12.23 breaths/min (range: 7.22 to 19.36 breaths/min). For speaking, tidal volume averaged 0.79 L (range: 0.35 to 1.24 L, representing 5.93% to 24.20 %VC) and breathing frequency averaged 20.00 breaths/min (range: 13.96 to 30.62 breaths/min). Differences between quiet breathing and speaking were significant for minute volume [$t(19) = 9.00$, $p < .01$], tidal volume [$t(19) = 2.48$, $p = .02$], and breathing frequency [$t(19) = 7.50$, $p < .01$].

During baseline quiet breathing, end-tidal P_{CO_2} averaged 37.61 mmHg for the group (range: 28.41 to 43.38 mmHg). During post-speaking quiet breathing, end-tidal P_{CO_2} was lower than mean baseline end-tidal P_{CO_2} in all but one subject (Subject 8). The magnitude of change in end-tidal P_{CO_2} from quiet breathing to post-speaking was –5.86 mmHg on average for the group (range: –14.00 to 1.75 mmHg). The difference between baseline quiet breathing end-tidal P_{CO_2} and post-speaking end-tidal P_{CO_2} was significant [$t(19) = 6.60$, $p < .01$].

The relationship of change in minute volume (from baseline quiet breathing to speaking) to change in end-tidal P_{CO_2} (from baseline quiet breathing to post-speaking quiet breathing) was examined for all speaking trials. This relationship, depicted in Figure 2, was characterized by a generally greater decrease in end-tidal P_{CO_2} with increased speaking-related minute volume. Calculation of the Pearson product moment correlation revealed a significant negative correlation between these two measures ($r = –.528$, $p < .001$).

Syllable-related measures for speaking varied across subjects, but were similar across trials for a given subject (individual subject means were calculated from the three trials combined). Group means for syllable-related measures were 16.11 sylls/breath group (range: 10.78 to 22.68 sylls/breath group), 5.11 sylls/s (range: 4.52 to 5.67 sylls/s), and 52.59 ml/syll (range: 35.17 to 77.19 ml/syll). Pearson product moment correlations were calculated to determine if these syllable-related measures were correlated with speaking-related changes

Table 2. Group means (and standard deviations) of ventilation-related measures for each of the three trials of speaking.

Measure	Trial 1	Trial 2	Trial 3
Minute volume (L/min)	14.01 (3.95)	13.29 (4.57)	13.99 (3.43)
Tidal volume (L)	0.80 (0.26)	0.77 (0.24)	0.80 (0.24)
Breathing frequency (breaths/min)	20.01 (4.77)	19.32 (6.10)	19.66 (4.53)
End-Tidal P_{CO_2} (mmHg)	35.86 (4.28)	35.56 (4.38)	35.06 (4.05)

Figure 1. Individual subject means and standard deviations for baseline quiet breathing (white bars) and speaking (cross-hatched bars) for minute volume (L/min), tidal volume (L), and breathing frequency (breaths/min). For end-tidal PCO_2 (mmHg), individual subject means and standard deviations are shown for baseline quiet breathing and individual subject means are shown for post-speaking quiet breathing that were obtained from breaths during the first 30 s immediately following speaking.

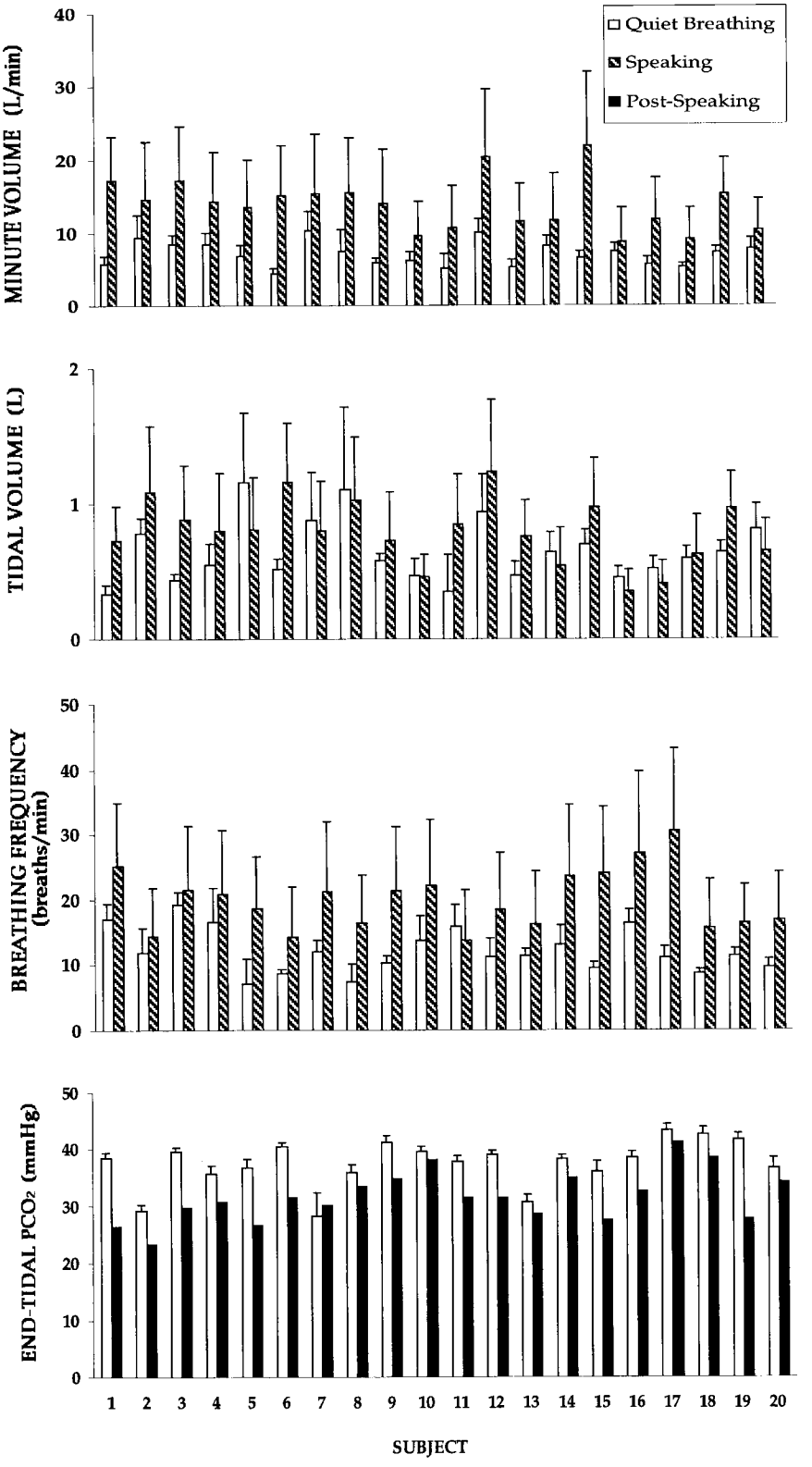
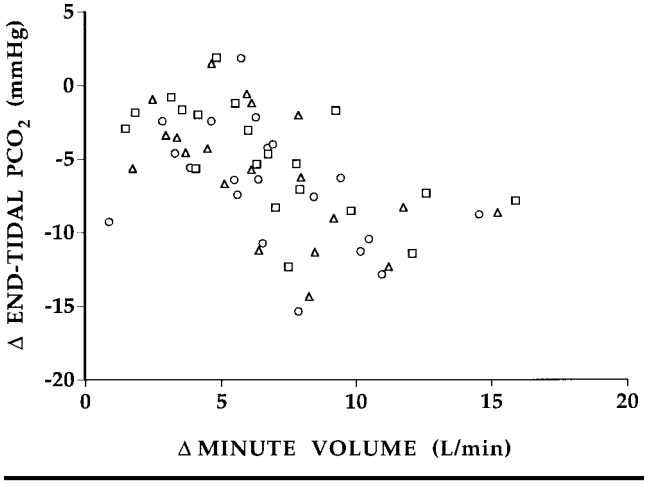


Figure 2. Graph depicting the relationship between change in minute volume (from baseline quiet breathing to speaking) and change in end-tidal PCO_2 (from baseline quiet breathing to the first 30 s post-speaking) for each of the three speaking trials for all 20 subjects. Squares, triangles, and circles represent trials 1, 2, and 3, respectively.

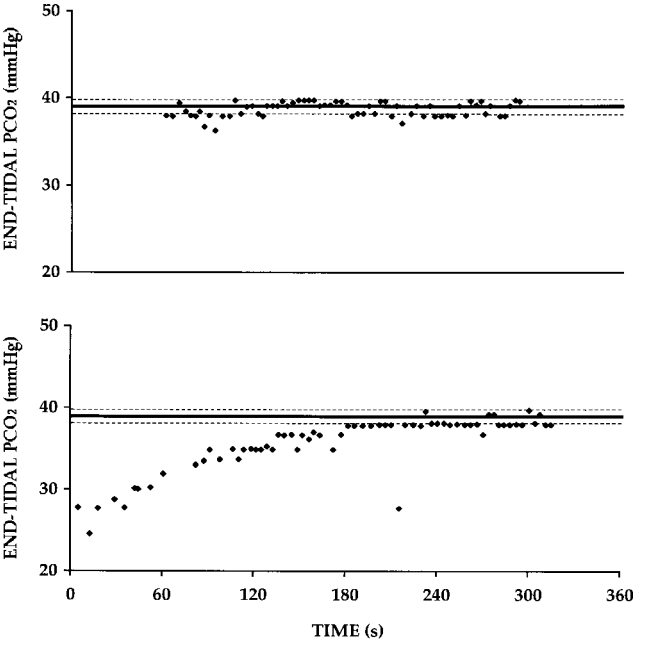


in ventilation. Correlation coefficients revealed that change in minute volume (from baseline quiet breathing to speaking) was not significantly correlated with sylls/breath group (r = .016, p = .903), but that it was significantly correlated with sylls/s (r = .380, p = .003) and ml/syll (r = .854, p < .001).

Following the (usually downward) shift in end-tidal P_{CO_2} that occurred after speaking, end-tidal P_{CO_2} eventually returned to baseline levels (i.e., within ±1 standard deviation of the mean). A typical example of breath-by-breath plots of end-tidal P_{CO_2} is shown in Figure 3. The upper panel of the figure shows end-tidal P_{CO_2} values for the 4-min baseline quiet breathing period, along with the corresponding mean (solid line) and standard deviation (dashed lines). The lower panel depicts end-tidal P_{CO_2} values during a period of post-speaking quiet breathing, which immediately followed a 10-min period of continuous speaking. Note that end-tidal P_{CO_2} was approximately 11 mmHg lower than baseline immediately following speaking and gradually increased to baseline levels. Examination of these end-tidal P_{CO_2} "recovery" periods from all trials of all subjects indicated that the median time to return to baseline was approximately 4 min, and the range was approximately 0.5 to 8.0 min.

Breath-by-breath measures of minute volume, tidal volume, and breathing frequency during post-speaking quiet breathing did not exhibit a "recovery" pattern as did measures of end-tidal P_{CO_2}. Rather, they were characterized by variability that exceeded variability during baseline quiet breathing. Coefficients of variation

Figure 3. Breath-by-breath plots of end-tidal PCO_2 (in mmHg) during baseline quiet breathing (upper graph) and during quiet breathing that immediately followed 10 min of continuous reading aloud (lower graph). Each filled circle represents the end-tidal PCO_2 for a single breath. The solid (and dashed lines) represent the mean (and standard deviation) obtained from baseline quiet breathing (Subject 1).



were larger for post-speaking quiet breathing than baseline quiet breathing in 98, 80, and 95% of the trials for tidal volume, breathing frequency, and minute volume, respectively. Variability usually appeared to be most pronounced during the first few breaths following cessation of speaking.

Discussion

Results of this study indicate that ventilation during speaking is greater than during quiet breathing. Discussion of these results begins with a critique of the method, followed by ventilation during quiet breathing and speaking, "recovery" of ventilation following speaking, and summary and clinical implications.

Critique of the Method

Certain design features of this study are relevant to generalization of results and comparisons with previous research. These are discussed below under the following headings: subjects, speaking task, and end-tidal P_{CO_2} measures.

Subjects

This study included 20 young men who met strict health and communication-related criteria, the largest group of homogeneous subjects included in a study of ventilation during speaking. A strength of this design feature is that the present set of data presumably provides a representative picture of ventilation behavior among healthy young men and includes a representative range of intersubject variability. A limitation of this design is that the present findings should not be generalized to other populations. For example, they should not be generalized to women, because women differ from men in hormonally related variables known to influence ventilation (Lyons, 1976; White, Douglas, Pickett, Weil, & Zwillich, 1983).

Although subject inclusion and exclusion criteria were quite stringent, subjects were not queried about their experience with yoga. Later, it was learned that one of the subjects (Subject 5) practiced yoga regularly. This subject was found to have unusual quiet breathing behavior in that his average tidal volume was larger and his average breathing frequency was lower than the other subjects. Perhaps most striking was his large breath-to-breath variability in tidal volume and breathing frequency, particularly during baseline quiet breathing. Interestingly, there was nothing unusual about his average minute volume or end-tidal P_{CO_2} nor his pattern of end-tidal P_{CO_2} recovery following the cessation of speaking (see "Recovery" of Ventilation After Speaking). Despite the fact that no apparent problems arose from the inclusion of this subject in the present study, it seems important to recognize that individuals who practice yoga (or other activities involving specialized breathing techniques) may be more aware of their breathing and may have a tendency to guide their quiet breathing consciously in ways that they believe are "preferred." Conscious breathing is much less likely to occur during speaking as the subject's attention is more apt to be focused on the written text (during reading aloud) or the formulation of the spoken message (during extemporaneous speaking).

Speaking Task

The present subjects were instructed to read aloud continuously for 10 min. This speaking task was designed to maximize the possibility that speaking would influence ventilation (by having subjects speak without stopping for a relatively long period). As a result, these subjects exhibited a near doubling in ventilation above quiet breathing levels.

The subjects of Bunn and Mead (1971) and Russell et al. (1998) also read aloud continuously for 5 min and 7 min, respectively. In contrast to the present subjects, their subjects increased ventilation only slightly above quiet breathing levels (i.e., less than 15%).[1] It is unclear why smaller increases in ventilation were elicited in these studies than in the present study. One possible reason could relate to differences in the nature of the reading material (e.g., average sentence length, grammatical structure). Meanock and Nicholls (1982) reported a 34% increase in ventilation above quiet breathing levels during a 5-min period of speaking; however, the nature of the speaking task was not specified.

The question arises as to whether or not ventilation increases (above quiet breathing levels) during a noncontinuous speaking activity such as conversational interchange. According to Warner et al. (1983), it does. They found that all of their subjects ventilated more during conversational speaking than at rest, with an average increase of approximately 40%. This increase is greater than that reported by Bunn and Mead (1971), Meanock and Nicholls (1982), and Russell et al. (1998) and smaller than that exhibited by the present subjects for continuous speaking.[2]

Although it appears that the speaking task may influence ventilation, the precise nature of that influence is not clear. Bunn and Mead (1971) have shown that phonetic content affects ventilation, with "high flow" reading material (i.e., containing a large proportion of voiceless consonants) eliciting substantially greater ventilation than "low flow" material (i.e., containing all voiced sounds). Whether or not variables such as average sentence length or grammatical structure have a similarly direct influence on ventilation has yet to be determined.

In the present study, ventilation was examined across three trials of the same speaking task and was found to be surprisingly consistent from trial to trial. Thus, speaking-related ventilation appears to remain stable for a given reading task, at least within a single recording session. Nevertheless, it is possible that trial-to-trial variability might be greater if subjects were studied on separate days, especially because lung volume events during reading have been shown to vary substantially across days (Winkworth, Davis, Ellis, & Adams, 1994).

End-Tidal P_{CO_2} Measures

The present method for obtaining speaking-related measures of end-tidal P_{CO_2} was different from those used

[1]The comparison data used here were those that were most analogous to the present data. From Bunn and Mead (1971), the data from the neutral reading passage were used. From Russell et al. (1998), the data from men speaking at a comfortable sound pressure level were used.

[2]Warner et al. (1983) reported increases in estimates of alveolar ventilation, whereas increases in total ventilation were reported by Bunn and Mead (1971), Meanock and Nicholls (1982), Russell et al. (1998), and the present study. Nevertheless, calculations of percentage increase should be generally comparable.

by previous investigators. In this study, speaking-related measures of end-tidal P_{CO_2} were made from nasally expired gas during periods of quiet breathing that came before and immediately after speaking. No such measures were obtained during periods of speaking. There were at least two reasons that P_{CO_2} was measured during quiet breathing only. One reason was that it was not possible to identify an adequate number of P_{CO_2} waveforms that met the measurement criterion (i.e., a plateau of at least 0.4 s). Another reason was that it was important to obtain these measures under conditions in which expirations were relatively consistent in duration, because duration can influence the measure of end-tidal P_{CO_2}. A longer-than-usual expiration (such as often occurs during speaking) tends to be associated with a higher-than-usual end-tidal P_{CO_2} because more time has elapsed for CO_2 to accumulate in the expired gas (Bunn & Mead, 1971).

End-tidal P_{CO_2} is a noninvasive measure that provides breath-to-breath estimates of alveolar ventilation in individuals with healthy lungs; however, the best indicator of overall alveolar ventilation is the partial pressure of CO_2 in arterial blood, or arterial P_{CO_2} (Kersten, 1989). When alveolar ventilation is lower than normal, less gas exchange occurs and a greater-than-normal amount of CO_2 builds up in arterial blood. Conversely, when alveolar ventilation is greater than normal, a lower-than-normal amount of CO_2 is found in arterial blood.

External validation of our method for estimating alveolar ventilation with measures of end-tidal P_{CO_2} might have been obtained with simultaneous measures of arterial P_{CO_2}. Nevertheless, this would have required arterial blood draws, a potentially dangerous procedure. Fortunately, there is a noninvasive method for obtaining valid estimates of arterial P_{CO_2} through the use of a transcutaneous sensor (Janssens, Howarth-Frey, Chevrolet, Abajo, & Rochat, 1998; Phan, Tremper, & Lee, 1987). This method was used to obtain external validation for the present method by making direct comparisons of end-tidal P_{CO_2} and transcutaneous P_{CO_2} recorded simultaneously. This was done in three subjects (none of whom were subjects in the study proper). End-tidal P_{CO_2} was measured as has been described for the study proper. Transcutaneous P_{CO_2} was sensed with a transcutaneous monitor (Radiometer TCM3, Copenhagen, Denmark). The sensor was fixed to the skin just caudal to the right clavicle at its midline. The speaking and breathing activities were the same as those in the study proper, except that the subjects performed only one trial of speaking instead of three (because ventilation had been shown to be consistent across trials). Results showed the same pattern of change (or nonchange) from baseline to post-speaking for the two sets of measures. Specifically, in one subject end-tidal P_{CO_2} dropped by approximately 4.5 mmHg and transcutaneous P_{CO_2} dropped by approximately 4.0 mmHg with speaking; in another subject, both measures dropped by approximately 2.5 mmHg with speaking; and in still another subject, there was essentially no change in either measure with speaking. The fact that transcutaneous P_{CO_2} exhibited the same patterns of change as did the end-tidal P_{CO_2} measures supports the validity of the present measurement method for estimating alveolar ventilation.

Whereas end-tidal P_{CO_2} was sampled during quiet breathing only in the present study, Bunn and Mead (1971) measured end-tidal P_{CO_2} by sampling nasally expired gas while their subjects were speaking, stating that in "most subjects there was sufficient nasal flow during speech so that acceptable plateaus of carbon dioxide could be obtained…" (p. 870). It is unclear why acceptable plateau pressures were found in the Bunn and Mead study but not in the present study, despite using the same method of sensing expired P_{CO_2}. Perhaps it is related to differences in instrumentation or differences in criteria for what was considered an "acceptable plateau." Nevertheless, Bunn and Mead found that end-tidal P_{CO_2} values associated with speaking were lower than those associated with quiet breathing. This finding, along with other observations, led Bunn and Mead to conclude that "…subjects tend to hyperventilate when they speak" (p. 872). Thus, the general findings of Bunn and Mead and those of the present study were the same, despite differences in measurement techniques.

Russell et al. (1998) used yet another method for obtaining speaking-related measures of end-tidal P_{CO_2}. Rather than using a nasal cannula to sense expired P_{CO_2}, they used a full face mask to capture expired gas from the oral and nasal airways. Speaking-related measures of end-tidal P_{CO_2} were obtained during speaking. Their results (for men speaking at comfortable loudness, the subject group/speaking task that most closely resembles those used in the present study) showed end-tidal P_{CO_2} during speaking to be higher on average than during quiet breathing. The authors concluded that their subjects "hypoventilated" during speaking.

The conclusions of Russell et al. (1998) contradict those of Bunn and Mead (1971) and those of the present study. It is not clear why. Nevertheless, it seems relevant to consider their use of a face mask for two reasons. First, despite the evidence that a face mask does not affect speech breathing (Huber, Stathopoulos, Bormann, & Johnson, 1998), there is ample documentation that a face mask can change quiet breathing behavior (e.g., Askanazi et al., 1980; Maxwell, Cover, & Hughes, 1985; Perez & Tobin, 1985). Second, using a face mask resulted in measures of end-tidal P_{CO_2} being obtained at the end of speech expirations. This is important because speech expirations are generally longer

than quiet breathing expirations (consistent with the values for expiratory time reported by Russell et al.),[3] and P_{CO_2} increases with expiratory duration (Bunn & Mead, 1971). Thus, end-tidal P_{CO_2} plateau pressures at the end of speech expirations might be expected to be higher than plateau pressures at the end of quiet breaths. To confirm this, a face mask was used to collect expired gas while recording P_{CO_2} during quiet breathing and speaking (personal observations, June 1998). These quiet breathing data showed the same pattern as observed in the present study proper—that is, end-tidal P_{CO_2} during quiet breathing was lower after speaking than before speaking (indicating an increase in ventilation associated with speaking). The data obtained during the act of speaking revealed that P_{CO_2} values were higher than those associated with quiet breathing and that the longer the duration of the spoken breath group, the higher the P_{CO_2}. Thus, the higher P_{CO_2} values obtained by Russell et al. appear to be a direct consequence of the fact that they made their measurements at the end of expirations that generally were longer in duration than those of quiet breathing. Perhaps if occasional quiet breaths (and/or short and breathy speech breaths) had been interspersed among speech breaths, lower levels of end-tidal P_{CO_2} might have been observed.

Ventilation During Quiet Breathing and Speaking

Ventilation-related behavior of the subjects during quiet breathing and connected speaking generally resembled those of other healthy young men (Bunn & Mead, 1971; Grimby, Bunn, & Mead, 1968; Haldane & Priestley, 1905; Hixon et al., 1973; Hoit & Hixon, 1986, 1987; Shea, Walter, Murphy, & Guz, 1987; Stathopoulos, Hoit, Hixon, Watson, & Solomon, 1991; Tobin, Mador, Guenther, Lodato, & Sackner, 1988). Nevertheless, substantial variability was observed across the present 20 subjects. Intersubject variability in ventilation-related measures has been reported by previous investigators (Haldane & Priestley, 1905; Shea et al., 1987; Winkworth et al., 1994). Variability in breathing behavior is not well-accounted for by physical characteristics such as height and weight (Haldane & Priestley, 1905; Shea et al., 1987), something that was also found to be true in the present group of subjects. Clear determinants of individual breathing patterns, whether neural or mechanical, have yet to be discovered (Shea & Guz, 1992).

All of the present subjects ventilated more when they spoke than when they breathed quietly. Speaking-related increases in ventilation were accompanied by decreases in end-tidal P_{CO_2} in all but one subject. If metabolism remains constant, there is a reciprocal relationship between arterial P_{CO_2} (i.e., the partial pressure of CO_2 in arterial blood) and alveolar ventilation (i.e., the amount of air that reaches the alveoli and participates in gas exchange) such that a doubling of alveolar ventilation causes a halving of arterial P_{CO_2}. Although minute volume (the present estimate of total ventilation) essentially doubled during speaking (from 7.15 L/min for quiet breathing to 13.94 L/min for speaking), the associated drop in end-tidal P_{CO_2} (the present estimate of arterial P_{CO_2}) was only about 15% (from 37.61 mmHg for quiet breathing to 31.75 mmHg for speaking). There are at least two potential reasons for this. One is that the act of speaking may involve an increase in metabolism (increasing the amounts of oxygen consumed and CO_2 produced). Another relates to the fact that nearly half the subjects used smaller tidal volumes and higher breathing frequencies for speaking compared to quiet breathing. This would have resulted in increased dead space and reduced the fraction of inspired air that participated in gas exchange. Support for this can be found in the fact that the subjects with smaller tidal volumes during speaking exhibited some of the smallest speaking-related changes in end-tidal P_{CO_2} of the group (see bottom of Figure 1, especially Subjects 7, 8, 10, 14, 17 and 20).

The magnitude of the speaking-related increase in minute volume varied substantially across subjects. Interestingly, its magnitude was found to be correlated with speaking rate (syll/s) and lung volume expenditure per syllable (ml/syll). Subjects who spoke more rapidly tended to have larger increases in minute volume (above quiet breathing levels); however, speaking rate accounted for less than 15% of the variance. By far the strongest correlation, accounting for approximately 73% of the variance, was between minute volume increase and lung volume expenditure per syllable. Subjects who expended more gas per syllable tended to exhibit greater increases in minute volume (above quiet breathing levels).

Although no formal perceptual analyses were included in this study, there is anecdotal evidence to suggest that speaking-related changes in ventilation may be perceptible in the acoustic speech signal. During data collection, while the subject was speaking, one or both investigators were able to predict with reasonable accuracy the change in end-tidal P_{CO_2} immediately following speaking. Although it cannot be certain what perceptual cues influenced these judgments, it seems reasonable to assume that one prominent cue might have

[3]Although the average expiratory time was longer than the average inspiratory time for all subject group/speaking task combinations (see Table 1, Russell et al., 1998, p. 243), the actual values do not agree with what has been reported by other investigators. For example, the average inspiratory time reported for men speaking at a comfortable sound pressure level was 1.46 s. This is nearly three-fold longer than those reported previously for inspirations during reading (Horii & Cooke, 1978; Solomon & Hixon, 1993). These values have been found to be in error and have been corrected (B. Russell, personal communication, January 1999).

been breathiness. Given the presumed link between perceived breathiness and measures of ml/syll and the strong correlation between ml/syll and ventilation found in this study, it may be that degree of breathiness is associated with magnitude of ventilation during speaking. If this is true, it may be possible to discern information about ventilation by attending to voice quality. This idea is now being tested formally.

"Recovery" of Ventilation After Speaking

Immediately after 10 min of continuous speaking, end-tidal P_{CO_2} was lower than it had been before speaking began (in all but one subject). End-tidal P_{CO_2} returned to its pre-speaking level, but the time course differed from subject to subject and from trial to trial. In general, the greater the reduction end-tidal P_{CO_2}, the longer it took to return to its baseline level. The rate at which end-tidal P_{CO_2} returned to baseline presumably depended on the duration of the preceding speaking-related hyperventilation (in this case, 10 min), which would have served to reduce the body's stores of CO_2 (in muscle, blood, and other tissues), the prevailing ventilation, and the rate of CO_2 production. The post-speaking end-tidal P_{CO_2} recovery curves generated by the present subjects are similar to those observed following voluntary (nonspeech) hyperventilation (Corfield, Morrell, & Guz, 1995).

Other quiet breathing variables also were affected by speaking. Perhaps most striking was the increased variability in breath-by-breath measures of minute volume, tidal volume, and breathing frequency. In most cases, variability was greatest for the initial 5 to 10 breaths following speaking. Changes in breathing patterns have been observed in humans immediately following voluntary hyperventilation (Corfield et al., 1995; Meah & Gardner, 1994) and have been ascribed to what has been termed "afterdischarge" or "short-term potentiation" (Eldridge, 1974; Fregosi, 1991). This phenomenon has been characterized by a continuation of increased ventilation for approximately 1 min following cessation of a hyperventilation stimulus and has been hypothesized to reflect persistence of activity due to reverberation within respiratory neural circuits (Eldridge, 1974). It would be interesting to determine if similar trends of ventilation change occur immediately following speaking.

Summary and Clinical Implications

This study confirmed previous observations that ventilation is greater during speaking than quiet breathing (Abel et al., 1987; Bunn & Mead, 1971; Meanock & Nicholls, 1982; Warner et al., 1983). Specifically, in nearly all cases, minute volume increased during speaking, resulting in lower-than-baseline end-tidal P_{CO_2} immediately following speaking, with relatively larger increases in minute volume generally being associated with a relatively greater reductions in end-tidal P_{CO_2}. This study also showed that (a) there can be a broad range of ventilation-related behaviors, even in subjects as homogeneous as the young men who participated in this study; (b) ventilation is consistent across repeated trials of the same speaking task; and (c) end-tidal P_{CO_2} and quiet breathing patterns are altered for up to several minutes following a period of continuous speaking. A strong positive correlation was found between speaking-related increases in ventilation (above baseline quiet breathing levels) and lung volume expenditures per syllable. This observation led to the speculation that judgments of voice quality, especially those related to breathiness, could provide perceptual indicators of the degree to which a person ventilates while speaking.

The results of this study are relevant to the respiratory-care practitioner responsible for the evaluation and management of clients with quiet (nonspeech) breathing dysfunction. Specifically, when measuring blood gases and monitoring quiet breathing patterns, the respiratory-care practitioner should be aware that prolonged speaking can increase ventilation and that a client's usual quiet breathing pattern can be perturbed for several minutes after speaking ceases.

The results of this study also are relevant to the speech-language pathologist responsible for the evaluation and management of clients with breathing-based speech disorders. The more that is known about the influence of speaking on ventilation in healthy individuals, the better the speech-language pathologist should be at recognizing abnormality in speech breathing function. For example, the degree to which ventilation increases during speaking may be exaggerated in individuals with respiratory compromise resulting from such conditions as asthma, emphysema, and sarcoidosis (Lee, Louden, Jacobson, & Stuebing, 1993; Louden, Lee, & Holcomb, 1988) or laryngeal compromise due to vocal fold paralysis (Miyazaki, Yamashita, Masuda, Yamamoto, & Komiyama, 1999). Although the most direct means of identifying abnormal function is through measures of ventilation, it may be that the speech-language pathologist can gain insight into a client's speaking-related ventilation by making judgments of voice quality. It may be that a breathy voice quality is a perceptual cue associated with excessive ventilation. By contrast, a strain-strangled voice quality may indicate abnormally reduced ventilation, such as might be found in clients with speech breathing disorders caused by restrictive respiratory conditions including spinal cord injury (Hixon, Putnam, & Sharp, 1983; Hoit, Banzett, Brown, & Loring, 1990;

Putnam & Hixon, 1983). This potential relation of voice quality to ventilation is speculative and offers fertile ground for future research.

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References

Abel, H., Mottau, B., Klubendorf, D., & Koepchen, H. (1987). Pattern of different components of the respiratory cycle and autonomic parameters during speech. In G. Sieck, S. Gandevia, & W. Cameron (Eds.), *Respiratory muscles and their neuromotor control* (pp. 109–113). New York: Alan R. Liss.

Askanazi, J., Silverberg, P., Foster, R., Hyman, A., Milic-Emili, J., & Kinney, J. (1980). Effects of respiratory apparatus on breathing pattern. *Journal of Applied Physiology, 48*, 577–580.

Bouhuys, A., Proctor, D., & Mead, J. (1966). Kinetic aspects of singing. *Journal of Applied Physiology, 21*, 483–496.

Bunn, J., & Mead, J. (1971). Control of ventilation during speech. *Journal of Applied Physiology, 31*, 870–872.

Corfield, D., Morrell, M., & Guz, A. (1995). The nature of breathing during hypocapnia in awake man. *Respiration Physiology, 101*, 145–159.

Eldridge, F. (1974). Central neural respiratory stimulatory effect of active respiration. *Journal of Applied Physiology, 37*, 723–735.

Fregosi, R. (1991). Short-term potentiation of breathing in humans. *Journal of Applied Physiology, 71*, 892–899.

Grimby, G., Bunn, J., & Mead, J. (1968). Relative contribution of rib cage and abdomen to ventilation during exercise. *Journal of Applied Physiology, 24*, 159–166.

Goldman-Eisler, F. (1955). Speech-breathing activity—A measure of tension and affect during interviews. *British Journal of Psychology, 29*, 53–63.

Haldane, J., & Priestley, J. (1905). The regulation of the lung-ventilation. *Journal of Physiology (London), 32*, 225–266.

Hixon, T., Goldman, M., & Mead, J. (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research, 16*, 78–115.

Hixon, T., Mead, J., & Goldman, M. (1976). Dynamics of the chest wall during speech production: Function of the thorax, rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Research, 19*, 297–356.

Hixon, T., Putnam, A., & Sharp, J. (1983). Speech production with flaccid paralysis of the rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Disorders, 48*, 315–327.

Hoit, J., Banzett, R., Brown, R., & Loring, S. (1990). Speech breathing in individuals with cervical spinal cord injury. *Journal of Speech and Hearing Research, 33*, 798–807.

Hoit, J., & Hixon, T. (1986). Body type and speech breathing. *Journal of Speech and Hearing Research, 29*, 313–324.

Hoit, J., & Hixon, T. (1987). Age and speech breathing. *Journal of Speech and Hearing Research, 30*, 351–366.

Horii, Y., & Cooke, P. (1978). Some airflow, volume, and duration characteristics of oral reading. *Journal of Speech and Hearing Research, 21*, 470–481.

Huber, J., Stathopoulos, E., Bormann, L., & Johnson, K. (1998). Effects of a circumferentially vented mask on breathing patterns of women as measured by respiratory kinematic techniques. *Journal of Speech, Language, and Hearing Research, 41*, 472–478.

Janssens, J., Howarth-Frey, C., Chevrolet, J., Abajo, B., & Rochat, T. (1998). Transcutaneous P_{CO_2} to monitor noninvasive mechanical ventilation in adults: Assessment of a new transcutaneous P_{CO_2} device. *Chest, 113*, 768–773.

Kersten, L. (1989). *Comprehensive respiratory nursing.* Philadelphia: W. B. Saunders.

Knudson, R., Lebowitz, M., Holberg, C., & Burrows, B. (1983). Changes in the normal maximal expiratory flow-volume curve with growth and aging. *American Review of Respiratory Disease, 127*, 725–734.

Lauber, P. (1989). *Voyagers from space: Meteors and meteorites*. New York: Thomas Y. Crowell.

Lee, L., Loudon, R., Jacobson, B., & Stuebing, R. (1993). Speech breathing in patients with lung disease. *American Review of Respiratory Disease, 147*, 1199–1206.

Loudon, R., Lee, L., & Holcomb, B. (1988). Volumes and breathing patterns during speech in healthy and asthmatic subjects. *Journal of Speech and Hearing Research, 31*, 219–227.

Lyons, H. (1976). Centrally acting hormones and respiration. *Pharmacology and Therapeutics: Part B: General and Systematic Pharmacology, 2*, 743–751.

Maxwell, D., Cover, D., & Hughes, J. (1985). Effect of respiratory apparatus on timing and depth of breathing in man. *Respiration Physiology, 61*, 255–264.

Meah, M., & Gardner, W. (1994). Post-hyperventilation apnoea in conscious humans. *Journal of Physiology, 477*, 527–538.

Meanock, C., & Nicholls, A. (1982). The effect of speech on the ventilatory response to carbon dioxide or to exercise. *Journal of Physiology, 325*, 16P–17P.

Miller, A. (1986). Prediction equations and "normal values." In A. Miller (Ed.), *Pulmonary function tests in clinical and occupational lung disease* (pp. 197–213). New York: Grune & Stratton.

Miyazaki, H., Yamashita, H., Masuda, T., Yamamoto, T., & Komiyama, S. (1999). Transcutaneous P_{CO_2} monitoring

in the evaluation of hyperventilation of patients with recurrent nerve paralysis. *European Archives of Oto-Rhino-Laryngology, 256 Suppl.*, S47–S50.

Perez, W., & Tobin, M. (1985). Separation factors responsible for change in breathing pattern induced by instrumentation. *Journal of Applied Physiology, 59*, 1515–1520.

Phan, C., Tremper, K., & Lee, S. (1987). Noninvasive monitoring of carbon dioxide: A comparison of the partial pressure of transcutaneous and end-tidal carbon dioxide with the partial pressure of arterial carbon dioxide. *Journal of Clinical Monitoring, 3*, 149–154.

Putnam, A., & Hixon, T. (1983). Respiratory kinematics in speakers with motor neuron disease. In M. McNeil, J. Rosenbek, & A. Aronson (Eds.), *The dysarthrias* (pp. 37–67). San Diego: College-Hill Press.

Reynolds, A., & Paivio, A. (1968). Cognitive and emotional determinants of speech. *Canadian Journal of Psychology, 22*, 164–175.

Russell, B., Cerny, F., & Stathopoulos, E. (1998). Effects of varied vocal intensity on ventilation and energy expenditure in women and men. *Journal of Speech, Language, and Hearing Research, 41*, 239–248.

Shea, S., & Guz, A. (1992). Personnalite ventilatoire—An overview. *Respiration Physiology, 87*, 275–291.

Shea, S., Walter, J., Murphy, K., & Guz, A. (1987). Evidence for individuality of breathing patterns in resting healthy man. *Respiration Physiology, 68*, 331–344.

Solomon, N., & Hixon, T. (1993). Speech breathing in Parkinson's disease. *Journal of Speech and Hearing Research, 36*, 294–310.

Stathopoulos, E., Hoit, J., Hixon, T., Watson, P., & Solomon, N. (1991). Respiratory and larnygeal function during whispering. *Journal of Speech and Hearing Research, 34*, 761–767.

Tobin, M., Mador, M., Guenther, S., Lodato, R., & Sackner, M. (1988). Variability of resting respiratory drive and timing in healthy subjects. *Journal of Applied Physiology, 65*, 309–317.

Warner, R., Waggener, T., & Kronauer, R. (1983). Synchronized cycles in ventilation and vocal activity during spontaneous conversational speech. *Journal of Applied Physiology, 54*, 1324–1334.

White, D., Douglas, N., Pickett, C., Weil, J., & Zwillich, C. (1983). Sexual influence on the control of breathing. *Journal of Applied Physiology, 54*, 874–879.

Winkworth, A., Davis, P., Ellis, E., & Adams, R. (1994). Variability and consistency in speech breathing during reading: Lung volumes, speech intensity, and linguistic factors. *Journal of Speech and Hearing Research, 37*, 535–556.

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Contact author: Jeannette D. Hoit, PhD, Department of Speech and Hearing Sciences, P.O. Box 210071, University of Arizona, Tucson, AZ 85721. Email: hoit@u.arizona.edu