Changes in speech breathing following cochlear implant in postlingually deafened adults

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Abstract

Three postlingually deafened adults who received cochlear implants read passages before and after their prostheses were activated. Their lung volumes were measured with an inductive plethysmograph that transduced the cross sectional areas of the chest and abdomen. The activation of the cochlear prostheses was followed in every case by a significant change in average airflow, which rose for two subjects with initially low flow rates and fell for one subject with a higher flow rate pre-implant [1].

1. Introduction

We have been studying speech breathing in late deafened adults as part of a larger project in which we examine physiological and acoustic properties of their speech while they perform a variety of speech tasks, before and after receiving electrical stimulation of the auditory nerve from a cochlear prosthesis. All three subjects became totally deaf in their twenties or thirties with profound bilateral sensorineural losses. Pre-implant they performed at chance levels on auditory tests of closed-set word recognition. Post-implant all three subjects improved in word and sentence recognition.

2. Procedure

In each session the subject read the elicitation passage three times at 20-minute intervals. Subjects F1 and F2 read the Rainbow Passage; M1 read "A Trip to the Zoo". There were two pre-stimulation baseline recording sessions.

Then the subjects began to receive electrical stimulation from their Ineraid multichannel cochlear implants, and additional recordings were made at intervals of approximately 1, 4, 12, and 24 weeks post-stimulation. The subjects did not receive auditory training or speech therapy. To obtain volumetric measures of speech breathing, we measured changes in the cross-sectional area of the rib cage and abdomen with an inductive plethysmograph (Respitrace). To compute the change in lung volume resulting from a respiratory maneuver, the two amplifier outputs from the plethysmograph are summed after weighting by correction factors. To determine the correct proportion of the two signals for a given recording session, the subject had to perform isovolume maneuvers at the beginning and again at the end of each session. To arrive at a scale factor for converting the summed volume signal to milliliters, the subject exhaled and inhaled into a plastic bag of calibrated volume. Amplified signals from the Respitrace and the microphone were recorded and low-pass filtered and digitized simultaneously. An operator labeled the beginning and end points of expiratory limbs while listening to the synchronized acoustic signal. The labeled events were automatically written into a file which was later accessed for calculating limb duration and limb initiation and termination levels in milliliters relative to FRC (tidal end respiration level).

3. Results

Figure 1 presents means of average airflow (left column) and volume of air expended per syllable (right) measured while postlingually deafened adults read passages three times in each of two sessions prior to receiving stimulation from a cochlear prosthesis, and in four (M1,F2) or six sessions (F1) following onset of stimulation. (The vertical lines show when the processor was turned on.) Each point is the mean for three passages. Vertical bars show +/- one standard deviation of the passage means around the session mean.

Figure 1: Average airflow (left column) and volume of air expended per syllable (right) measured while postlingually deafened adults read passages three times in each of two sessions prior to receiving stimulation from a cochlear prosthesis, and in four (M1,F2) or six sessions (F1) following onset of stimulation.
from cochlear prostheses and in four (M1,F2) or six (F1) sessions subsequently. M1, hearing-impaired since birth, averaged initially 18.1 mL/sec of airflow. After two weeks’ stimulation from his prosthesis (onset indicated by vertical line) M1 had reduced his average airflow by 15% (third session). On the average, M1’s flow rates, after his processor was turned on, were 17% lower than before stimulation (f(1,2) = 2.26, p < .05). In sessions 1 and 2, M1 expended an average of 68.2 mL/sec. After activation of the processor, the volume of air expended fell on the average over four sessions to 46 mL/sec, a decrement of 33% (F(1,2) = 2.42, p < .01). Prior to implant, M1 ended his expiratory limbs 79.6 mL below FRC. This is characteristic of congenitally deaf speakers. Two weeks after activation of the processor, M1’s termination level fluctuated around FRC (F(1,2) = 2.30, p < .05). It appears that M1 used his newfound economy of average airflow when reading following implant mostly to desist drawing on expiratory reserve volume (the volume below FRC).

Subject F1, a female, initially expended air during reading at abnormally low rates, averaging 92.0 mL/sec. Following the onset of stimulation, her average airflow increased gradually and irregularly, attaining 144.0 mL/sec after 85 weeks. The mean airflow in all recordings following activation of the processor was 104.3 mL/sec, an increase of 13.4% over the two baseline sessions (F(1,2) = 23.9, p < .05). We observed informally that F1’s voice quality has also changed: before stimulation, it was harsh and loud; now it is much softer. The volume of air that F1 expended per syllable increased following activation of the processor by 7.9%, from 23.9 to 25.8 mL (F(1,2) = 33.2, p < .05).

Subject F2 also started out with abnormally low rates of average airflow while reading. Following stimulation, her average airflow increased 20.2%, from an average of 125.8 mL/sec for the two baseline sessions to 151.2 mL/sec for recordings pooled over the four sessions post-implant (F(1,2) = 537.4, p < .01).

4. DISCUSSION

Figure 2 plots mean average airflow (left) and volume per syllable expended before and after activation of the implant. Insofar as our three speakers are representative of postlingually deafened adults, it appears that the effects of total sensorineural hearing loss in adulthood include anomalies in the management of speech breathing and that these may involve either an expenditure of too much air or of too little. Once the speakers received some auditory input from their cochlear prostheses, in every case they modified their speech breathing in the direction of normality. Significant changes were observed in average airflow (M1,F1,F2), in volume of air expended per syllable (M1,F1), and in speech termination levels (M1). Some of the changes in the acoustic correlates of speech that are associated with sudden hearing loss may be mediated by

abnormal patterns of speech respiration and laryngeal control of the breath stream. Similarly, some of the acoustic changes that take place when partial self-hearing is restored by cochlear prosthesis may be mediated by a normalization of breath stream mechanisms such as observed in this study. Improper laryngeal valving is a prime suspect in the search for mechanisms underlying the excessive air expenditure of some late deafened speakers and the inadequate air volumes and flow rates of others.

REFERENCE