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Speech motor learning in profoundly deaf adults

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Speech production, like other sensorimotor behaviors, relies on multiple sensory inputs—audition, proprioceptive inputs from muscle spindles and cutaneous inputs from mechanoreceptors in the skin and soft tissues of the vocal tract. However, the capacity for intelligible speech by deaf speakers suggests that somatosensory input alone may contribute to speech motor control and perhaps even to speech learning. We assessed speech motor learning in cochlear implant recipients who were tested with their implants turned off. A robotic device was used to alter somatosensory feedback by displacing the jaw during speech. We found that implant subjects progressively adapted to the mechanical perturbation with training. Moreover, the corrections that we observed were for movement deviations that were exceedingly small, on the order of millimeters, indicating that speakers have precise somatosensory expectations. Speech motor learning is substantially dependent on somatosensory input.

One of the puzzles of human language is that individuals who become deaf as adults remain capable of producing quite intelligible speech for many years in the absence of auditory input[1–3]. This ability suggests that speech production is substantially dependent on nonauditory sensory information and particularly on afferent input from the somatosensory system. Previous studies that have sought to identify a somatosensory basis for speech motor function have done so in the presence of auditory inputs[4–12] and therefore any effects that were observed may have resulted from the presence of the auditory signal. Here we found that somatosensory input on its own may underlie speech production and speech motor learning. We studied speech learning in cochlear implant recipients, who we tested with their implants turned off. We assessed speech learning by using a robotic device that applied forces that displaced the jaw and altered somatosensory feedback during speech. We found that implant subjects progressively corrected their speech movements to offset errors in the motion path of the jaw even in the absence of auditory input. Indeed, the levels of adaptation that we observed were comparable for implant subjects and normal-hearing control subjects. This indicates that speech learning is substantially dependent on somatosensory feedback. Speech production must be understood both as an auditory[13–15] and a somatosensory task[11,12,16].

RESULTS

Five post-lingually deaf adults participated in the study (**Fig. 1a**). These subjects had profound hearing loss in both ears (average hearing loss, 101 dB). All but one had received a cochlear implant (**Fig. 1b**). The hearing loss for the subjects with cochlear implants was sensorineural in origin; the remaining subject, who wore a hearing aid, had a mixed sensorineural and conductive hearing loss (see Methods). Six age-matched control subjects (average hearing loss, 13 dB) had hearing that was typical of their age range (**Fig. 1b**). During the experimental session, a robotic device applied a mechanical load to the jaw as the subject repeated aloud test utterances that were chosen randomly from a set of four (saw, say, sass and sane) and displayed on a computer monitor. The mechanical load was velocity dependent[17,18] and acted to displace the jaw in a protrusion direction, altering somatosensory, but not auditory, feedback[11,12]. Subjects were trained over the course of 300 utterances. Sensorimotor learning was evaluated using a measure of movement curvature to quantify adaptation. Curvature was measured at the point of maximum jaw-lowering velocity and was calculated as jaw protrusion divided by the magnitude of jaw elevation at this point. The hearing-impaired subjects were trained with their implant or hearing aid turned off, whereas control subjects had full hearing during training.

We plotted jaw trajectories in speech for a representative implant subject and a normal-hearing control subject (**Fig. 2a**,**b**). In both cases, movements were straight in the absence of load, the jaw was displaced in a protrusion direction when the load was first applied, curvature decreased with training and there was a small aftereffect following the unexpected removal of load. Movements for the implant subject under no load conditions were similar regardless of whether the implant was on or off. We examined movement curvature for an implant subject for individual trials over the entire course of the experiment (**Fig. 3a**). As before (**Fig. 2a**), the values of curvature were low in the null condition, increased with the introduction of load and then progressively decreased with training.

Kinematic and acoustical tests of adaptation were conducted quantitatively on a per subject basis using analysis of variance (ANOVA) followed by Tukey's honestly significantly different (HSD) *post hoc* tests. Subjects showed similar kinematic patterns in both the implant and control groups. In the implant group, adaptation was observed in all five subjects, as indicated by a significant decrease in curvature over the training period ($P < 0.01$ for all subjects; **Fig. 3b**). Only four of six

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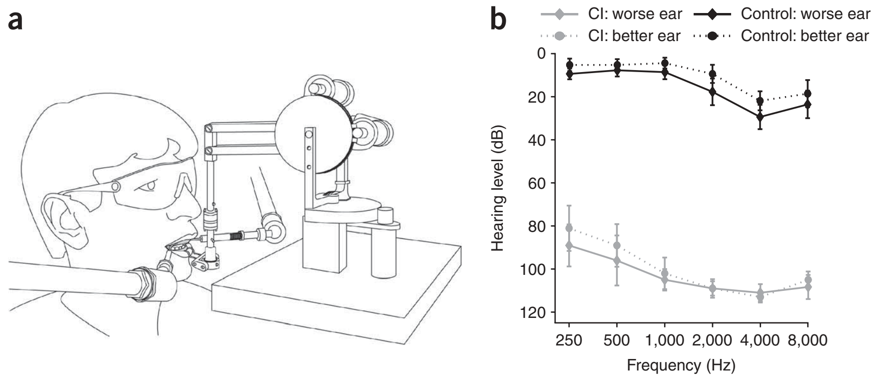

Figure 1 Experimental set-up and audiogram. (**a**) A robotic device delivered a velocity-dependent load to the jaw. (**b**) Pure-tone hearing thresholds. Cochlear implant subjects (CI, shown in gray) had a severe to profound hearing loss. Control subjects (black) had hearing levels that were typical for their age.

control subjects adapted to the load ($P < 0.01$; **Fig. 3c**). The amount of adaptation was assessed on a per subject basis by computing the reduction in curvature over the course of training as a proportion of the curvature resulting from the introduction of load. A value of 1.0 indicates complete adaptation. Adaptation, averaged across subjects and test words, was 0.20 ± 0.06 (mean ± s.e.m.) for the implant group and 0.19 ± 0.04 for the control group. Adaptation was thus comparable for the two groups ($P > 0.93$, *t*-test), suggesting that, for the set of four utterances that we tested, auditory feedback is not necessary for adaptation to load. Subjects' response to the sudden removal of the load following training was variable. Two of the four control subjects that adapted to load showed reliable aftereffects by *post hoc* tests, as did three of five implant subjects ($P < 0.01$).

In individuals with normal hearing, the adaptation observed here could have been driven by somatosensory or auditory feedback, or by the two in combination: somatosensory feedback is altered because the load alters the movement path of the jaw and changes somatosensory input, and auditory feedback may also change because the load might affect speech acoustics by altering the shape of the vocal tract. Because subjects in the implant group adapted with the implant turned off, auditory input does not seem to be necessary for speech learning, at least in post-lingually deaf adults.

To evaluate the presence of auditory cues for adaptation that might have been used by the normal-hearing control subjects, we assessed acoustical changes in the speech signal over the course of training. Acoustical effects related to the application of load were evaluated by computing the first and second formant frequencies of the vowel immediately following the initial consonant s in each of the test utterances. We plotted the raw acoustical signal for the test utterance saw and the associated first and second formants of the speech spectrogram (**Fig. 4a**). We chose these particular vowels for acoustical analysis because their production coincided with the opening phase of jaw movement during which the force field was maximum. We reasoned that the load's effect, if any, on the speech signal should be most evident at this point. The acoustical data included for analysis were for only those subjects who adapted to load.

We assessed the acoustical effects quantitatively on both a between subjects basis and for each subject separately for both implant and control subjects (**Fig. 4b,c**). We focused on potential effects of the load's introduction, possible changes with learning and changes resulting from the unexpected removal of load in the aftereffect trials. A repeated-measures ANOVA produced few statistically reliable acoustical effects over the course of learning. As expected, the acoustical patterns differed for the different test words ($P < 0.01$ for both formants). However, the acoustical effects were similar for implant and control subjects ($P > 0.69$ and $P > 0.58$ for the first and second formants, respectively). In one case, for the utterance sass, there was a reliable increase in the first formant frequency over the course of training ($P < 0.05$). However, we found no other statistically reliable differences in either the first or second formant frequency with the introduction of load, from the start to the end of training or on the sudden removal of load. We repeated these analyses for the implant and control groups separately and obtained no reliable differences in formant frequencies between these various phases of the experiment.

We also assessed possible acoustical and kinematic differences in the implant group in the null condition with and without the implant on. For this test, we used the last half of the no-load trials with the implant on and an equal number of trials at the end of the no-load phase with the implant off. Typically this amounted to 40 or 50 utterances in each condition. A repeated-measures ANOVA (across subjects and test utterances) found no differences in either the first or the second formant frequencies ($P > 0.89$ and $P > 0.93$, respectively) or in movement curvature ($P > 0.97$). This indicates that any systematic changes in production patterns resulting from switching off the implant had been eliminated before the force field was introduced.

We used tests of correlation to assess the extent to which changes in movement curvature over the course of learning were related to first and second formant values. Tests were conducted for each subject using the mean curvature and the mean formant frequency in each block over the course of the experiment. Separate correlations were computed for the first and second formant frequencies. There was little evidence that changes in movement curvature with learning were mirrored in the acoustical domain. In the case of the first formant, hearing-impaired

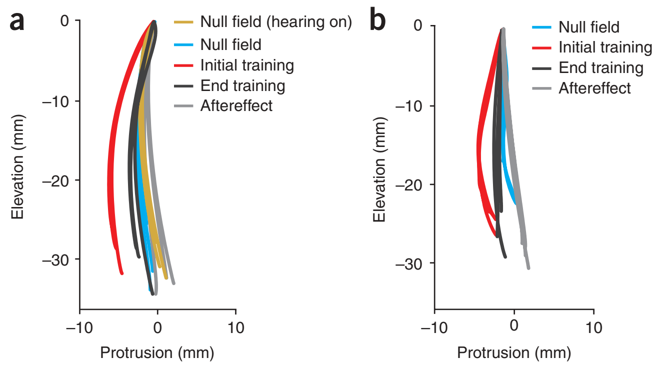

Figure 2 Sagittal plane jaw-movement paths. Speech motor learning in implant recipients, who were trained with their implants turned off, was similar to that of normal-hearing control subjects. (**a**) For implant subjects, jaw paths were straight in the absence of load (gold, implant turned on; cyan, implant turned off). The jaw was deflected in the protrusion direction when the load came on (red). After training, movement curvature decreased (black). When the load was switched off unexpectedly at the end of training, there was a small aftereffect (gray). (**b**) Control subjects showed a similar pattern. Color codes are the same as those used in **a**. In all cases, individual movements are shown.

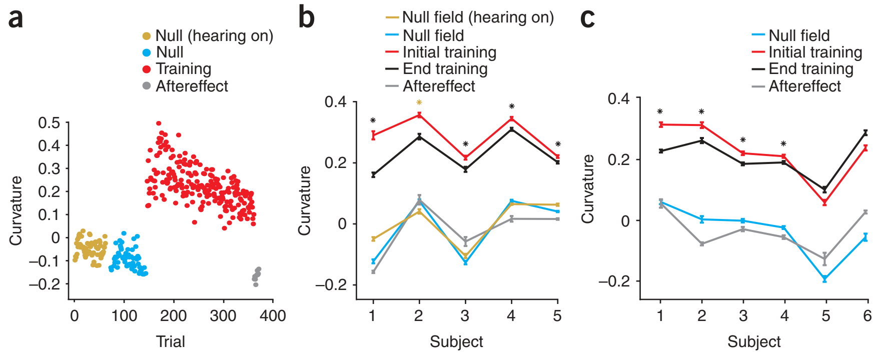

Figure 3 Adaptation patterns in implant and control subjects. (**a**) Scatter plot showing learning for a representative implant subject. The ordinate depicts movement curvature and the abscissa gives trial number. Curvature was low during null trials (gold and cyan), increased with the introduction of the load and then decreased over the course of training (red). A small aftereffect was seen when the load was switched off (gray). (**b**) Significant adaptation was observed in all implant subjects. The figure shows mean curvature (± s.e.m.) during various phases of the experiment. Curvature increased with the introduction of load (red) and decreased reliably with training (black). Asterisks (*) designate statistically reliable adaptation ($P < 0.01$). The subject with mixed hearing loss is shown with a gold star. (**c**) Significant adaptation was also observed in 4 out of 6 normal-hearing control subjects.

subjects showed a mean correlation of –0.12 ± 0.10 (mean ± s.e.m.) between formant value and curvature. The corresponding correlations for the control group were –0.01 ± 0.01. For the second formant, these same correlations yielded 0.02 ± 0.09 and 0.002 ± 0.02, respectively. Thus, there is no indication that changes in movement curvature with learning have any effects on the formant frequencies of the associated speech.

The adaptation seen in implant subjects may have been caused, in part, by changes in somatosensory and/or kinematic precision that have occurred to compensate for the auditory loss. As already noted, all of our implant subjects showed statistically reliable adaptation, whereas only two-thirds of the normal-hearing control subjects (4 out of 6) had similar patterns. We looked for differences in the kinematic and acoustical characteristics of the two groups under null conditions. We examined the first two formants and associated values of jaw protrusion and elevation (**Fig. 5a,b**). Using ANOVA, we tested for differences in implant and control subjects in acoustical and kinematic parameters in the absence of load (implant turned off). We found no systematic differences between these groups in first ($P > 0.95$) or second formant frequency ($P > 0.97$) or in jaw protrusion ($P > 0.53$) or elevation ($P > 0.11$). We further assessed possible differences between implant and control subjects in acoustical and kinematic precision by computing their respective coefficients of variation, which are measures of variability normalized by the mean. We plotted the coefficients of variation of the first two formants and the coefficients of variation of protrusion and elevation (**Fig. 5c,d**). No differences in the coefficients of variation between implant and control subjects were found for either the first or the second formant frequency ($P > 0.68$ and $P > 0.31$, respectively) or for horizontal jaw position ($P > 0.95$). For vertical jaw position, control subjects were found to have a marginally greater coefficient of variation ($P = 0.06$).

DISCUSSION

We have examined speech motor learning in post-lingually deaf adults who were tested with their implants turned off. Implant subjects showed significant adaptation, comparable to that observed in control subjects, to a mechanical load that acted to displace the jaw and alter somatosensory feedback. Neither group of subjects showed measurable acoustical change as a consequence of the load. These data are consistent with the idea that speech motor learning is reliant on somatosensory feedback and that even subtle changes in movement prompt corrective adjustments. The somatosensory guidance of speech movements by deaf speakers may underlie the capacity for intelligible speech following hearing loss.

It is important to consider the possibility that deaf individuals speak intelligibly in the absence of auditory input because they use stored motor programs, in effect a sequence of motor commands, which are executed without sensory feedback. Our findings indicate that speech trajectory representations cannot be encoded simply as motor

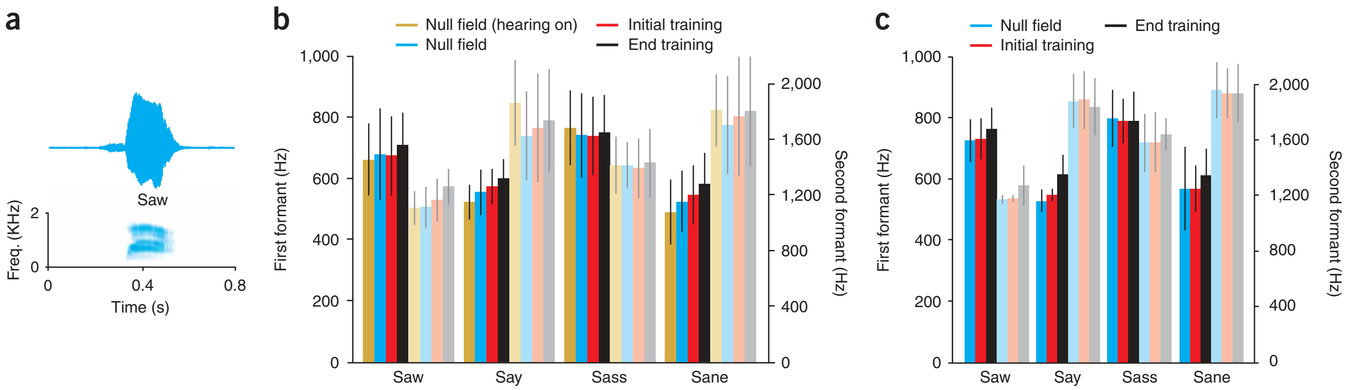

Figure 4 There were no systematic acoustical effects associated with force field learning. (**a**) Top, the raw acoustical waveform for the word saw. Bottom, the first two formants of the corresponding spectrogram. (**b**) The load had little effect on the acoustics of the implanted subjects. The first formants of vowels were computed under no load conditions (gold and cyan), at the introduction of the load (red) and at the end of training (black). The second formants are shown in pale colors, with the frequency scale shown on the right. (**c**) The load had little effect on the acoustics of the normal-hearing control subjects. The first and the second formants of vowels were computed under no load conditions (cyan), at the introduction of the load (red) and at the end of training (black). In all cases ± s.e.m. is shown.

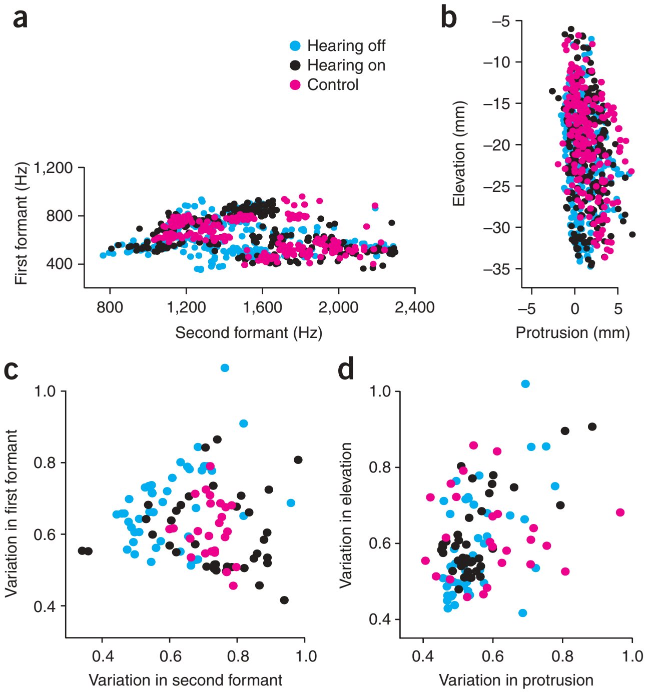

Figure 5 Kinematic and acoustical variability for implant (with implant on and off) and control subjects. (**a**) Acoustical variability was similar for implant and control subjects. Representative examples across all subjects and utterances of first and second formant frequencies are shown for no load trials. (**b**) Both subject groups have comparable variation in jaw kinematics. Representative examples of vertical and horizontal jaw position during the null trials. Position values are computed at the point at which formant values are evaluated. (**c**) Implant and control subjects have similar coefficients of variation for formant frequencies in the absence of load. (**d**) Corresponding coefficients of variation for jaw position. The individual data points give null condition values of the coefficient of variation for each utterance and each subject separately.

commands or else we would not observe adaptation to mechanical perturbations. The compensation observed here cannot be acoustic, as there is limited acoustic feedback that might regulate the adaptation. Accordingly, compensation in our study must involve a somatosensory trajectory representation and somatosensory feedback. Moreover, both are probably used on a routine basis in the production of speech and speech motor learning.

The adaptation shown by the implant group may reflect a heightened sensitivity to somatosensory input as a consequence of hearing loss, but it might also reflect the normal role of somatosensory inputs in determining speech movements. Our data provide some support for both possibilities. The fact that the compensation observed here is similar for implant and control subjects suggests that the implant group is no more sensitive to somatosensory change than subjects with normal hearing. However, all subjects in the implant group showed adaptation in comparison with the more typical two-thirds proportion that we observed in the control group[11,12,19]. This difference would argue in favor of the theory that somatosensory sensitivity is improved in at least some individuals with late-onset hearing loss.

We were able to dissociate the role of auditory and somatosensory feedback by assessing speech learning in deaf adults who receive no auditory information during speech. Both here and in other studies, we achieved a comparable dissociation by applying loads that alter jaw movement and, hence, change somatosensory feedback without any perceptible change to the acoustics[11]. The small acoustical effects are presumably the results of the nonrigid coupling between the jaw and the acoustically critical tongue surface. Moreover, the perturbations are quite small and change the length of the vocal tract by millimeters at most. The expected acoustical effect is therefore rather limited.

The degree to which subjects compensate for load is comparable in implant subjects and in age-matched controls. Adaptation was incomplete in both cases; on average, there was about a 20% reduction in movement error over the course of training. However, partial adaptation is typical of studies of speech motor learning, both with mechanical loads and altered acoustical feedback[11–15,19], and may reflect the imprecision of articulatory targets and the possibility for inter-articulator trade-offs in the achievement of auditory goals. In studies of speech motor learning with mechanical loads, there is typically somewhat greater adaptation[11,12]. The age of subjects may be a determining factor, as our subjects were considerably older than those of previous studies. This may have contributed to a greater tolerance for movement errors and thus reduced adaptation.

As was already mentioned, only two-thirds of our control subjects showed evidence of adaptation. Indeed, adaptation rates in studies of altered auditory feedback are in a similar range[19]. Although this remains to be tested, the observed adaptation rates may reflect an individual's reliance on auditory versus somatosensory feedback. Subjects in our study who failed to adapt may rely less on somatosensory function and more on auditory feedback, whereas subjects who failed to adapt in studies of altered auditory feedback may be more reliant on somatosensory function and less on auditory feedback[19].

We should comment on the possibility of auditory feedback in subjects in our implant group. Auditory feedback ordinarily reaches the cochlea on the basis of air-conducted and also bone-conducted signals. The consensus opinion is that the basilar membrane simply sums them[20,21]. Individuals that have a profound sensorineural hearing loss, such as the four implant recipients tested in this experiment, should not hear signals that reach the cochlea, regardless of whether those signals arrive by air or bone conduction. The fifth subject in the implant group, who had a mixed hearing loss with both sensorineural and conductive components, may receive some low-frequency bone-conducted auditory input when they speak, but given their substantial sensorineural hearing loss, they probably experience a much-attenuated signal at best[22]. It is worth noting that this subject's speech-adaptation patterns were similar to those of the other subjects in the implant group and to those of control subjects with normal hearing. We believe that the similarity of speech learning over the spectrum of hearing loss underscores the conclusion that speech production and speech motor learning are not strictly tied to auditory input.

Our finding that implant and control subjects achieved a comparable level of adaptation has implications for multisensory integration in speech. Speech production typically involves integration of auditory and somatosensory inputs. In subjects with normal hearing, inputs from each modality contribute to the error information that drives adaptation. The simplest possibility is that the nervous system linearly sums error information to achieve a composite measure of total sensory error[23,24]. For implant subjects, particularly in the context of our study, where testing occurred shortly after the implant was turned off, we found that subjects could rapidly place reliance on somatosensory input to achieve adaptation and could seemingly discount the auditory channel. The weighting of sensory inputs is not fixed and indeed it seems possible to quickly alter the weighting if necessary.

METHODS

Subjects and tasks. We tested 11 subjects in total (**Fig. 1b**); five of them (age 63.8 ± 8.5 years, two females and three males) had an average hearing loss of 101 dB for air-conducted sound and the other six age-matched control subjects (age 55.6 ± 4.7 years, two females and four males) had normal hearing (average loss of 13 dB). All of the hearing-impaired subjects had post-lingually acquired deafness and had lost their hearing gradually. All but one had received a cochlear implant in the ear with the worse hearing. The one remaining subject wore a hearing aid. The onset of hearing loss ranged from age 6 to 56 with a mean onset age of 33.2. On average, subjects had received their implant 2.5 years before participating in this study. The Institutional Review Board of McGill University approved the experimental protocols.

We evaluated subjects in the implant group for the possibility of conductive hearing loss (failure of signal transmission through the tympanic membrane or middle ear) by assessing auditory thresholds to bone-conducted sound and by tympanometry. We found no evidence of a conductive hearing loss in any of the four subjects with cochlear implants. In other words, their deafness was entirely sensorineural in origin. For these subjects, bone-conduction auditory thresholds measured at frequencies from 250–4,000 Hz showed a hearing loss that exceeded the limits of the audiometer for bone-conduction testing (in the range of 65-dB hearing loss to a bone-conducted signal).

The subject that used a hearing aid had a mixed hearing loss with both a conductive and a sensorineural component. This particular subject was profoundly deaf to air-conducted sound. Their hearing loss for the right ear averaged 105 dB. The left ear hearing loss was 96 dB. The bone-conduction thresholds measured at 250, 500, 1000, 2,000 and 4,000 Hz showed a hearing loss of 30, 40, 35, 55 and 50 dB, respectively. The less-severe hearing loss for bone-conducted sound at lower frequencies was consistent with the finding that the bone-conduction transfer function peaks between 700 and 1,200 Hz (ref. 22). It is thus possible that this subject would hear the low frequencies of his own voice through bone conduction during speech production. However, because of his substantial sensorineural hearing loss, any transmission through the cochlea would be attenuated.

The task was to repeat a single word test utterance that was displayed on a computer monitor while a robotic device delivered a velocity-dependent load to the jaw that acted in the protrusion direction. The experiment was carried out in blocks of 12 utterances each. On each trial, the test utterance was chosen randomly from a set of four words, saw, say, sass and sane, and each of the test utterances in the set was presented three times, on average, in a block of 12 utterances. The test words were selected so that in each case the fricative consonant s was followed by a vowel or a dipthong. Production of the consonant s involves a precise jaw position near to closure. The vowels and dipthongs were chosen to give large-amplitude jaw movements and high force levels. The display of the word was controlled manually by the experimenter, which introduced a delay of 1–2 s between the test utterances.

The first 3–5 blocks were recorded under null or no-load conditions. The implants or hearing aid were left on for this first set of trials, which constituted the hearing-on null phase of the experiment. Normal-hearing control subjects were tested in a similar number of blocks in the null condition. The subject's implant was then turned off and stayed off for the remainder of the experiment. After the implant was first turned off, there was a waiting period of approximately 10 min before subsequent testing began. At this point another 10–15 blocks were recorded under null conditions for the implant group. This constituted a hearing-off null phase. We recorded this large number of no-load trials after turning off the implant because there are rapid changes in the speech formant structure when the implant is first turned off[25,26], and we wanted to ensure that the production pattern had stabilized before training commenced. The next 25 blocks, approximately 300 repetitions of the test utterances, were recorded with the load on and constituted the training phase. Following the training phase, the load was unexpectedly turned off and one block of 'catch' trials was recorded in the absence of load.

Experimental procedures. A computer-controlled robotic device (Phantom Premium 1.0, Sensable Technologies) was used to deliver a load to the lower jaw. The robotic device was connected to a custom-made acrylic-metal dental appliance via a magnesium-titanium rotary connector that offered fully unconstrained movement of the jaw in the absence of external load. The dental appliance was attached to the buccal surface of the mandibular teeth with a dental adhesive (Iso-Dent, Ellman International). A force/torque sensor (ATI Nano-17, ATI Industrial Automation) was mounted at the tip of the robotic device to measure the resistive force applied by the subjects in opposition to the load. The subject's head was restrained during the experiment by connecting a second dental appliance that was glued to the maxillary teeth to an external frame that consisted of a set of articulated metal arms. The metal arms were locked in place throughout the experimental session.

Jaw movement was recorded in three dimensions at a rate of 1 kHz and the data were digitally low-pass filtered offline at 8 Hz. The subject's voice was recorded using a unidirectional microphone (Sennheiser). The acoustical signal was low-pass analog filtered at 22 KHz and digitally sampled at 44 KHz.

The robot applied a mechanical load to the jaw that resulted in jaw protrusion. The load varied with the absolute vertical velocity of the jaw and was governed by $F = k|v|$, where F is the load in newtons, k is a scaling coefficient and v is the jaw velocity in mm s^{-1}. The scaling coefficient was chosen to have a value of between 0.6–0.8, with a higher coefficient being used for subjects who spoke more slowly and vice versa. The maximum load was capped at 7.0 N, however. Jaw velocity estimates for purposes of load application were obtained in real time by numerically differentiating jaw position values obtained from the robot encoders. The computed velocity signal was low-pass filtered using a first-order Butterworth filter with a cut-off frequency of 2 Hz. The smoothed velocity profile was used to generate the protrusion load online.

Data analysis. A measure of path curvature, the ratio of protrusion to elevation at the peak vertical velocity of the jaw, was computed for each repetition of the test utterance. The jaw opening movement was used for analysis. We scored the movement start and end at 10% of peak vertical velocity. The first two trials in each training block were excluded from analysis to guard against the possibility that subjects initially stiffened up at the onset of force application.

We assessed adaptation by computing the mean curvature for the first and last 25% of the force field training trials. This gave approximately 50 movements in each case. A similarly computed measure of null condition performance was obtained by taking the mean of the last 50% of trials in the null condition blocks (both with implant on and off). Statistical assessments of adaptation were conducted using null blocks and initial and final training blocks. The effect on movement curvature of the unexpected removal of the load following training was evaluated relative to the null condition baseline level. This was done by subtracting the mean of the null condition values from the aftereffect block[11]. The first five trials in the aftereffect block were used for this analysis. A t-test was conducted to determine whether the mean of the normalized aftereffect values was negative.

Acoustical effects were quantified by computing the first and second formant frequencies of the vowels. An interval of approximately 100 ms, which contained the steady-state portion of the vowel, was selected manually on a per trial basis. The formants in this interval were computed using a formant-tracking algorithm that was based on the standard linear predictive coding procedures implemented in Matlab. We used a 25-ms analysis window. The median of the formant estimates in the interval was used for subsequent analyses.

Statistical analysis. The main statistical analyses were conducted using ANOVA followed by Tukey's HSD *post hoc* tests.

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AUTHOR CONTRIBUTIONS

S.M.N. and D.J.O. designed the experiments and wrote the manuscript. S.M.N. conducted the experiments and analyzed the data.

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