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**Abstract:** Cerebellar degeneration (CD) has deleterious effects on speech motor behavior. Recently, a dissociation between feedback and feedforward control of speaking was observed in CD: Whereas CD patients exhibited reduced adaptation across trials to consistent formant feedback alterations, they showed enhanced within-trial compensation for unpredictable formant feedback perturbations. In this study, it was found that CD patients exhibit abnormally increased within-trial vocal compensation responses to unpredictable pitch feedback perturbations. Taken together with recent findings, the results indicate that CD is associated with a general hypersensitivity to auditory feedback during speaking.


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## 1. Introduction

The cerebellum is thought to play a role in many aspects of movement coordination, including sequencing, timing, motor programming, inverse modeling, and sensory prediction (Manto *et al.,* 2012). However, its role in the control of speech has received less attention than other types of movement control. Lesion and functional neuroimaging studies have shown that the cerebellum is a crucial part of the speech motor control network (Ackermann, 2008; Bohland and Guenther, 2006; Ghosh *et al.,* 2008). Neuroimaging studies have also shown increased cerebellar activation in response to both auditory (Tourville *et al.,* 2008) and somatosensory (Golfinopoulos *et al.,* 2011) perturbations of speech. Nevertheless, the specific functional role of the cerebellum in speech production remains unclear.

Speech abnormalities are prevalent in patients with cerebellar degeneration (CD) (Duffy, 2005; Spencer and Slocomb, 2007), including changes in voice production, such as harshness and vocal tremor (Lechtenberg and Gilman, 1978). Examining speech of such patients provides a unique opportunity to assess functional hypotheses about the cerebellum's role in speech motor control.

 

Current models of speech motor control emphasize the importance of both predictive (feedforward) and reactive (feedback) processes. Given the rapidity and complexity of speech, current models of speech motor control emphasize the importance of the role of internal, predictive forward models in motor control of speech (Houde and Nagarajan, 2011; Tourville and Guenther, 2011). Nevertheless, current models also include feedback control processes because even skilled speakers are sensitive to feedback information. Evidence for this comes from studies showing that alterations in auditory feedback result in rapid, on-line changes to speech production (Burnett *et al.,* 1998; Houde and Jordan, 1998; Lee, 1950; Purcell and Munhall, 2006).

For non-speech movements, CD patients show profound deficits in predictive (feedforward) motor control: Across a broad range of tasks involving reaching and locomotion, these patients exhibit a marked impairment in adapting to a consistent perturbation (Day *et al.,* 1998; Kawato, 1999; Manto *et al.,* 2012; Morton and Bastian, 2006; Shadmehr and Krakauer, 2008; Wolpert *et al.,* 1998). Although less well studied, reactive (feedback) motor control mechanisms appear to be relatively intact in this population (Rost *et al.,* 2005). In a recent study of speech motor control, we observed a similar pattern when we manipulated vowel formants. CD patients were impaired in adapting their feedforward control system relative to controls, exhibiting an attenuated anticipatory response when F1 was consistently shifted down by 150 Hz on each trial (Parrell *et al.,* 2017). In contrast, when F1 was randomly shifted up or down by 150 Hz on each trial (thus precluding anticipatory responses) the patients demonstrated hypersensitivity to sensory feedback, producing larger, within-trial compensatory responses than controls (Parrell *et al.,* 2017).

In the current study, we extend our prior studies to the control of pitch. An important question remaining to be answered is whether the hypersensitivity to formant feedback is indicative of hypersensitivity to auditory feedback in general during speaking. Here, we address this question by testing the hypothesis that CD patients will also show hypersensitivity to pitch feedback perturbations during vocalization.

## 2. Methods

### *2.1 Participants*

Sixteen patients (10 male) with cerebellar degeneration (CD), and 11 healthy (7 male) aged matched controls participated in the experiment. The average age of the patient group was 50 years (+/− 12) and the age matched heathy control group was 51 years (+/− 11). The patients had cerebellar atrophy with heterogeneous diagnoses, including various types of spinocerebellar ataxia (SCA): SCA2 (2), SCA3 (2), SCA5 (1), SCA6 (2), SCA7 (1), SCA8 (2), and unknown/idiopathic cerebellar atrophy (6). No CD patients reported any history of neurological damage or disorder apart from cerebellar atrophy. Apart from typical high-frequency hearing loss associated with aging, none of the participants reported a history of speech or hearing problems. All of the participants signed informed consent approved by the University of California, Berkeley or University of California, San Francisco.

### *2.2 Apparatus and procedure*

The pitch-perturbation experiment consisted of two successive 74-trial sessions. Each trial began with a visual cue (a clearly visible dot on the screen) presented on a computer screen in front of the participant. The participants phonated the vowel /a/ as long as the visual cue persisted on the screen, such that the total vocal duration was ~2.4 s. The dot was followed by a 2.5-s blank screen period before the dot reappeared for the next trial. Every 15 trials, the participants were cued by the video screen to take a break, which the experimenter ended after confirming with the participant that he/she was ready to continue.

In each trial, onset of the participant's phonation triggered a brief perturbation of the pitch of his/her feedback after a randomly jittered delay (200–500 ms). The perturbation was either upwards or downwards by 100 cents (1/12 of an octave) [see Figs. 1(A) and 1(B)], with the direction randomly determined on each trial (without replacement). The perturbation was implemented with a real-time speech feedback alteration program. The input was the participant's phonation, as picked up by a head-mounted microphone (AKG Pro Audio C520), and the output from the computer was fed back into circumaural headphones (Beyerdynamic DT 770 PRO). The feedback alteration program decomposed incoming speech into pitch and spectral envelope features which could be separately altered before being recombined to synthesize the speech output to the earphones [for details, see Katseff *et al.* (2012)]. This process incurred a feedback delay of 12 ms. The auditory input through the earphones was

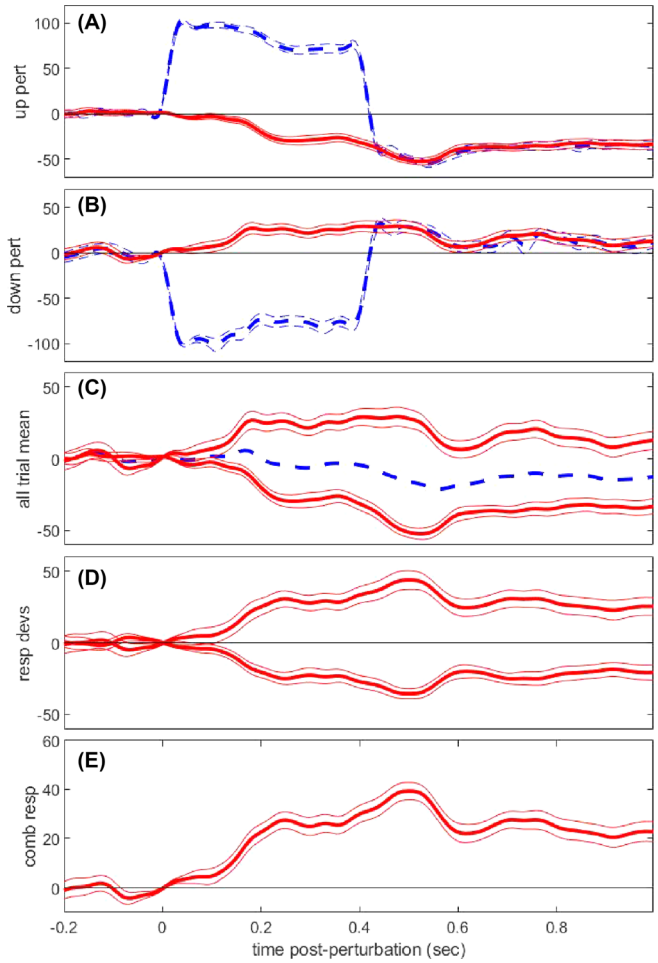


Fig. 1. (Color online) Pitch track processing steps. Plots show how the pitch data for an example control subject were processed. (A) Response to the up (+100 cent) pitch feedback perturbation, aligned to perturbation onset at 0 s. Dashed line (with flanking +/− s.e.m. lines) shows mean pitch of subject's feedback on the up perturbation trials, in cents relative to reference interval (+/− 50 ms around perturbation onset), exhibiting the +100 cent, 400 ms perturbation applied to the subject's auditory feedback on these trials. Solid line (again with flanking +/− s.e.m. lines) shows mean pitch of subject's produced pitch response to upward (+100 cent) pitch feedback perturbations, in cents relative to the same reference interval. (B) Mean response (with flanking +/− s.e.m.) to downward (−100 cent) pitch feedback perturbations. (C) Mean responses to the up and down pitch feedback perturbations (solid lines with flanking +/− s.e.m. lines) overlaid together, showing also the mean across all trials (both up and down perturbation trials) as dashed line. (D) Mean responses to the up and down pitch feedback perturbation trials (solid lines with flanking +/− s.e.m. lines), expressed as deviations from the mean across all trials. (E) Mean (+/− s.e.m.) of all the trials expressed as response deviations show in plot (D), combined by flipping the sign of the responses to the upward pitch feedback perturbations. In all plots, vertical axis is measured in cents.

adjusted to around 85 db sound pressure level (SPL), which tended to be louder than the bone conduction feedback of their actual voice.

### *2.3 Data processing and analysis*

Audio data of both the participants' speech and the pitch-altered feedback were recorded at 11.025 KHz. For each participant, the raw audio data for each trial was first analyzed into a time-course of the pitch signal using an autocorrelation-based pitch tracking method (Parsons, 1987). Analysis intervals stretching from 200 ms prior to onset of the pitch perturbation to 1000 ms following onset were extracted from the pitch time-course. Trials with pitch tracking errors or incomplete utterances within the analysis interval were excluded from further analysis. For the remaining trials, pitch tracks were converted from Hz to cents by the formula: cents (t) = 1200 * log2 [Hz (t) / HzRef], where Hz (t) refers to the pitch at time t, and t = 0 s is the time of perturbation onset. In this formula, cents (t) is a measure of pitch change at time t relative to a reference pitch (HzRef), which for each trial was calculated as the mean pitch over a reference interval spanning 50 ms prior to perturbation onset to 50 ms after perturbation onset.

Figure 1 shows how the resulting pitch track data were processed. Many participants had a net decrease in pitch over the time-course of the trial. This made the responses to up perturbations (for which compensation entails a decrease in pitch) appear to be larger than the responses to down perturbations. This bias can be seen in an example control subject's data in Fig. 1, plots (A) and (B). To correct for this bias, for each participant, the pitch track in cents for each trial was expressed as deviations from the mean pitch track, averaged across all trials (i.e., including responses to both the up and down pitch feedback perturbations). This process is shown in Fig. 1, plots (C) and (D). This approach removed the influence of any overall changes in produced pitch over the time-course of each trial, with the caveat that it also averaged across any true asymmetries in response to the up versus down perturbation.

The final step in processing each participant's response pitch tracks was to combine responses to both upward and downward perturbations into a single dataset. To generate a combined perturbation response data set for each participant, the deviations from the mean time-course in response to the upward perturbations were flipped (i.e., negate the cents deviation values of the time-course), and then the flipped trials were added to the data set of deviations from the mean time-course in response to the downward perturbations. The result of this combining process is shown in Fig. 1, plot (E). Trials were excluded either due to pitch tracking errors within the analysis interval or due to incomplete utterances. For the patient group, out of a total of 148 trials, the average number of trials included in the analysis per subject was 76 ($\pm$40), and for the control group subjects, the average number of included trials was 120 ($\pm$20).

For statistical analysis, the pitch contour for each individual trial was divided into 20 ms bins and the pitch value was averaged within these bins. For each bin, the distribution of trial means for patients and the distribution of trial means for controls were tested for significant difference from zero, via t test, and tested for significant difference from each other, via one-way analysis of variance (ANOVA). To control for inflated type I error rate, Bonferroni thresholds were applied resulting in a p-value significance threshold of 0.001. For comparisons of the peak perturbation response between patients and controls, a linear mixed-effects model was used to examine group differences, with trial as a covariate.

We also examined the variability of subjects' responses, both within-trial and across-trial, to determine whether patients were more variable than controls, and how responding to the pitch feedback perturbation affected that variability. For each subject, mean pitch track variability was measured as the standard deviation in cents both within each trial and across all trials in the baseline interval (the 200 ms prior to pitch feedback perturbation) and in an interval around the time of peak compensation (400–600 ms post perturbation). The resulting mean within- and across-trial variability data were then subject to separate repeated measures ANOVAs, with group (patient, control) and interval (pre/post perturbation) as factors.

## 3. Results

Figure 2 shows the response to the pitch perturbation for the patients (dotted blue lines) and controls (solid red lines). Note that in this analysis, compensation is always in the positive direction. There are similar morphological features in the two functions. For both groups, a change in pitch in response to the perturbation becomes evident around 160 ms after the onset of the perturbation, and this change persists for the remainder of the utterance. The groups first begin to differ from each other at 200 ms post-perturbation and remain different from each other until 639 ms post-perturbation. The peak of the mean control group response occurred at 526 ms post-perturbation and was 20.3 cents, while the peak of the mean patient group response occurred at 514 ms post-perturbation and was 39.6 cents. While the time of peak response occurrence for the two groups was quite close, the magnitude of the peak patient response was much greater than the peak control response ($p = 1.05 \times 10^{-15}$).

Patients' pitch tracks were significantly more variable than controls, both in the variability measured within trials [patients mean: 46.34 (2.92), standard error of the mean (s.e.m.) in parentheses, control mean: 22.39 (3.17); $p = 1.4851 \times 10^{-6}$] and across trials [patients mean: 39.52 (2.11), control mean: 19.19 (2.29); $p = 5.4643 \times 10^{-8}$]. For both groups, onset of the feedback perturbation increased pitch track variability, both in the variability measured within trials [pre-perturbation mean: 28.59 (3.05), post-perturbation mean: 40.1326 (3.05); $p = 0.0104$] and across trials [pre-perturbation mean: 22.03 (2.20), post-perturbation mean: 36.68 (2.20); $p = 2.4946 \times 10^{-5}$]. However, patients' variability was no more affected by perturbation onset than it was for controls

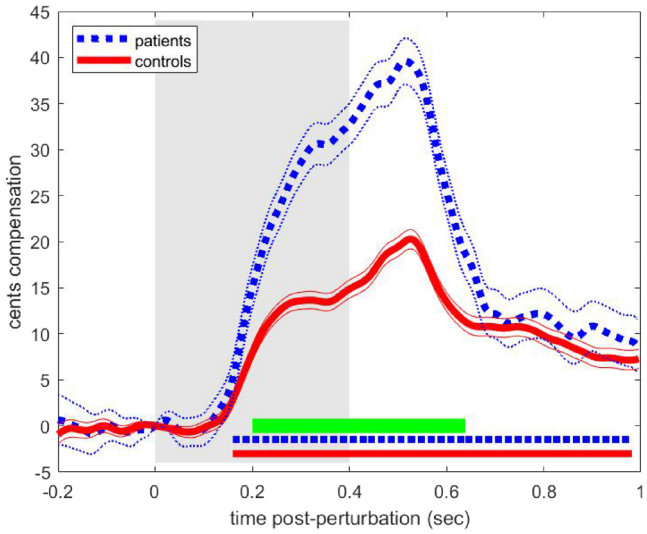


Fig. 2. (Color online) Pitch feedback perturbation responses. The result of the combined trial analysis for patients (dotted lines) and controls (solid lines). For each group's plot, thick lines indicate the mean responses across trials of each group and the flanking thin lines indicate +/− s.e.m. Shaded interval shows onset and duration of the pitch feedback perturbations. The horizontal bars below the group plot lines indicate results of a binned interval analysis of group response differences (see text). Solid and dotted bars just above the *x* axis indicate when the control and patient groups, respectively, significantly differed from zero. Thick bar above the other two bars indicates when the two groups' responses significantly differed from each other. For all tests, to control for inflated type I error rate, Bonferroni thresholds were applied resulting in a p-value significance threshold of 0.001.

(group by interval interaction: $p = 0.4159$ for within-trial variability and $p = 0.1370$ for across-trial variability).

## 4. Discussion

The key finding in the current experiment is that responses to transient pitch feedback perturbations by patients with cerebellar damage (CD) are significantly greater than that seen in controls. Morphological features of the groups' responses were in many ways the same: the two groups showed similar response onset times, peak response times, and similar levels of post-response persistence [i.e., what other studies have referred to as pitch rebound error (Behroozmand *et al.*, 2015)]. These response features are similar to what we have found in other studies of pitch perturbation responses (Demopoulos *et al.,* 2018; Naunheim *et al.,* 2018; Ranasinghe *et al.,* 2017; Subramaniam *et al.*, 2018). Furthermore, both within-trial and across-trial pitch variability was greater in the CD patients than in controls, which is consistent with prior studies of speech variability in CD (Kent *et al.*, 1997). Most notably, however, the pitch perturbation response was significantly enhanced in the CD group when compared to controls.

The current study had several limitations. First, for many study participants, we were unable to conduct rigorous hearing tests. Nevertheless, we note that the onset latency of the pitch compensation response was identical between the control and patient cohorts. We believe this onset latency reflects the auditory processing stage of the pitch compensation and a lack of any difference strongly suggests that basic auditory processing was not different between our patient and control cohorts. Second, our experiment design lacked no-perturbation catch trials, which meant that in our data analysis, the influence of any overall changes in produced pitch unrelated to the feedback perturbations was removed by expressing the pitch track of each trial as a deviation from the mean pitch track, averaged across all trials (i.e., including responses to both the up and down pitch feedback perturbations). This design choice not only reduced our experiment time but also increased the sensitivity of our study to detect any differences in pitch compensation between our study cohorts. However, this increased sensitivity came with a trade-off in specificity: Our design did not allow us to examine any asymmetry in the pitch compensation response between upwards vs downwards pitch shift. Having established a statistically significant difference in the pitch compensation response in CD patients, a follow-up study is warranted to examine if there are any asymmetries in the abnormal pitch compensation responses of CD patients.

Nevertheless, taken together with the previously-reported finding of greater response to F1 perturbations in CD patients (Parrell *et al.,* 2017), the result of this experiment suggests CD results in a general increase in sensitivity to auditory speech feedback. In fact, the effect for pitch is quite strong, with CD patients producing compensatory pitch changes that were nearly twice the magnitude of control responses. This is a much larger difference than what we saw in our previous study on F1 formant perturbation responses.

What could account for the difference? One technical difference between the two studies is the duration of the feedback perturbation. The formant perturbation experiment used whole-trial perturbations—on each trial F1 was shifted as soon as the utterance onset was detected and remained present for the entire utterance production. In the present pitch perturbation experiment, the perturbation was only present for a 400 ms interval in the middle of the produced utterance. However, it is unlikely that this variable explains the difference between the effect size for the two speech features. Prior studies have shown that responses to utterance-initial feedback perturbations tend to be larger than responses to mid-utterance perturbations (Hawco and Jones, 2009). Another possible explanation for the difference is that the control of pitch may, in general, be more dependent on auditory feedback than the control of formants. This can be seen in the effects of post-lingual deafness, in which the control of pitch and loudness degrades rapidly after hearing loss, while the articulation of speech, as conveyed by formants, remains intelligible for decades (Cowie and Douglas-Cowie, 1992). Another possible source of differences between F1 and pitch feedback sensitivity may arise from non-linearities in the vocal tract transfer function (Stevens, 1999). These non-linearities would mean that the relationship between articulatory muscle changes and F1 changes would be different from the relationship between laryngeal muscle changes and pitch changes.

A hypersensitivity to auditory feedback across speech features may help account for some of the instabilities in speech observed in individuals with cerebellar dysarthria (Kent *et al.,* 1997). Excessive reliance on sensory feedback is inherently unstable, given delays associated with processing the feedback (Houde and Nagarajan, 2011). This instability could result in increased variability during production. This explanation of the effects of hypersensitivity to sensory feedback is consistent with some effects of blocking feedback seen in CD in non-speech motor tasks: For example, blocking vision of the arm reduces endpoint reach variability in CD (Day *et al.,* 1998).

Why would CD be associated with hypersensitivity to sensory feedback? One possibility is suggested by the fact that feedback processing in pitch production includes processing both auditory and somatosensory feedback, as suggested by the fact that modulation of somatosensory feedback by local anesthesia enhances the pitch perturbation response (Larson *et al.*, 2008). In this study, we have only shown hypersensitivity to auditory feedback. If the cerebellum is preferentially involved in processing somatosensory feedback as some models of speech production suggest (Hickok, 2012), then damage to the cerebellum might favor a shift to a greater reliance on auditory feedback. Another possibility arises from an alternative perspective that considers the cerebellum's role in feedforward control.

As mentioned earlier, current models of speech motor control emphasize that speech production relies on a combination of feedback and feedforward control (Houde and Nagarajan, 2011; Tourville and Guenther, 2011). In these models, feedback control is implemented via a feedback correction system based on applying a gain to an auditory feedback prediction error, i.e., the difference between incoming auditory feedback and predicted auditory feedback. Therefore, the observed hypersensitivity to auditory feedback would be implemented in such models as an increase in the gain on auditory feedback prediction errors. Such a gain increase on sensory errors, increasing reliance on feedback control in CD patients would be expected if, as discussed above, the cerebellum plays a critical role in feedforward control. Damage to the cerebellum impairs feedforward control, which could favor a shift to greater reliance on sensory feedback control.

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