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RESEARCH ARTICLE

Control of Movement

Movement variability can be modulated in speech production

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Abstract

Although movement variability is often attributed to unwanted noise in the motor system, recent work has demonstrated that variability may be actively controlled. To date, research on regulation of motor variability has relied on relatively simple, laboratory-specific reaching tasks. It is not clear how these results translate to complex, well-practiced tasks. Here, we test how variability is regulated during speech production, a complex, highly overpracticed, and natural motor behavior that relies on auditory and somatosensory feedback. Specifically, in a series of four experiments, we assessed the effects of auditory feedback manipulations that modulate perceived speech variability, shifting every production either toward (inward pushing) or away from (outward pushing) the center of the distribution for each vowel. Participants exposed to the inward-pushing perturbation (*experiment 1*) increased produced variability while the perturbation was applied as well as after it was removed. Unexpectedly, the outward-pushing perturbation (*experiment 2*) also increased produced variability during exposure, but variability returned to near-baseline levels when the perturbation was removed. Outward-pushing perturbations failed to reduce participants' produced variability both with larger perturbation magnitude (*experiment 3*) and after their variability had increased above baseline levels as a result of the inward-pushing perturbation (*experiment 4*). Simulations of the applied perturbations using a state-space model of motor behavior suggest that the increases in produced variability in response to the two types of perturbations may arise through distinct mechanisms. Together, these results suggest that motor variability is actively monitored and can be modulated even in complex and well-practiced behaviors such as speech.

NEW & NOTEWORTHY By implementing a novel auditory feedback perturbation that modulates participants' perceived trial-to-trial variability without affecting their overall mean behavior, we show that variability in the speech motor system can be modulated. By assaying speech production, we expand our current understanding of variability to a well-practiced, complex behavior outside of the limb control system. Our results additionally highlight the need to incorporate the active control of variability in models of speech motor control.

auditory perturbation; error sensitivity; motor variability; sensorimotor adaptation; speech motor control

INTRODUCTION

No matter how hard we practice, it is virtually impossible to generate exactly the same movement twice. Such variation in performance across repetitions of the same movements, or motor variability, is widely believed to be an inevitable consequence of noise in the nervous system, arising from stochastic events presented across all scales of brain activity, from the single-cell level to complex network dynamics (1–3). Indeed, many current theories of motor behavior rely on this assumption and posit that the motor system aims to minimize the detrimental effects of "motor noise" on motor task performance (4–6).

However, recent work in reaching has demonstrated that variability is not always treated as unwanted "noise" to be reduced by the motor system but may be more actively controlled. Repeated exposure to position- or velocity-dependent force fields during reaching has been shown to selectively increase task-relevant variability, potentially to facilitate more efficient future learning (7). Conversely, task-relevant variability can also be reduced when needed in some behaviors: participants exposed to a visual perturbation that magnified

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the horizontal displacement of the hand away from the midline during point-to-point reaching movements reduced their variability in this dimension (8). These results suggest that variability is not simply noise (i.e., random, unwanted variation or fluctuation that interferes with the signal) but can also be an important part of the signal itself that controls the motor movement (9).

Although variability poses a fundamental problem for motor control, studies on regulation of motor variability are comparatively sparse and, to date, have relied principally on relatively simple, laboratory-specific planar arm reaching tasks. Here, we aimed to test how variability is regulated in speech production, a complex, well-practiced task controlled via nonvisual sensory feedback. In speech, vowel sounds are defined by vocal tract resonances, known as formants, that reflect different vocal tract sizes and shapes. The first two formant frequencies, F1 and F2, correspond roughly to the height and front-back position of the tongue body, respectively, and are sufficient to disambiguate vowel sounds (10). Previous work has shown that speakers are sensitive to real-time alterations to their auditory formant feedback during both single-word (11, 12) and sentence-level (13, 14) productions. For example, if F1 for the vowel in "head" is increased, it sounds closer to the vowel in "had." After receiving such altered auditory feedback, speakers learn to change their formant productions in the opposite direction to the shift applied (i.e., lowering their F1 by changing the tongue height). However, previous studies have typically examined formant shifts that are applied in a consistent direction, regardless of the produced vowel formants, with the consequence that the mean formant values participants hear are altered while their formant variability remains unchanged. Here, we implement a novel auditory perturbation that shifts the frequency of vowel formants in a nonuniform manner, such that the mean formant values remain unchanged while trial-to-trial variability is either increased or decreased (Fig. 1).

By altering participants' perceived trial-to-trial variability without affecting their overall mean behavior, we can test whether variability in speech production is actively monitored and regulated. In a series of four experiments, we assessed the effects of manipulations that both increase and decrease the perceived variability of participants' speech behavior. We predicted that a perturbation that reduces perceived variability would lead to increases in produced variability, as participants would be free to be less precise in their production without negatively affecting their perceived accuracy. Conversely, we expected that a perturbation that increases perceived variability would have the opposite effect (i.e., lead to decreases in produced variability). Surprisingly, we found that both types of perturbation caused participants to increase their produced variability, whereas only the perturbation that increases perceived variability affected a behavioral measure of error sensitivity. Simulations of the applied perturbations using a well-established state-space model of sensorimotor learning suggest that the increases in produced variability in response to the two types of perturbations may arise through distinct mechanisms: an increase in controlled variability in response to the perturbation that reduces perceived variability and an increase in sensitivity to auditory errors in response to the perturbation that increases perceived variability. All together, these results suggest that motor variability is actively monitored and can be modulated even in complex and well-practiced behaviors such as speech.

METHODS

Participants

Eighty-seven native speakers of American English between the ages of 18 and 66 yr, with no reported history of hearing loss or neurological disorders, took part in the study (*experiment 1*: $N = 24$, 19–60 yr, mean 28.8 ± 12.3 yr, 18/6 females/males; *experiment 2*: $N = 22$, 18–50 yr, mean 25.9 ± 9.7 yr, 14/8 females/males; *experiment 3*: $N = 21$, 19–66 yr, mean 33.3 ± 13.6 yr, 12/9 females/males; *experiment 4*: $N = 20$, 18–55 yr, mean 28 ± 12.9 yr, 15/5 females/males). Participants provided written informed consent and were compensated either monetarily or with course credit for their participation. The Institutional Review Board of the University of Wisconsin-Madison approved the experimental protocol.

Given that the primary goal of our statistical analysis was to examine how variability changed across different phases using repeated-measures analyses of variance (ANOVAs), a group of 24 participants (*experiment 1*) was deemed sufficient in terms of sensitivity to detect a significant ($\alpha = 5\%$) change in produced variability (in response to an inward-pushing perturbation) of at least medium effect size (Cohen's $f = 0.25$), should one occur, 8 times out of 10 (G*power 3.1). We then halved the effect size of variability change observed in *experiment 1* [partial eta squared (η_p^2) = 0.25] and determined that even at 90% power a sample size of 17 would be sufficient to detect a significant ($\alpha = 5\%$) change in produced variability. Given that we were also interested in other measurements (e.g., centering), we recruited at least 20 participants for each of the following experiments.

Apparatus

Participants were exposed to a real-time perturbation of the first and second vowel formants (F1/F2) designed to affect the perceived variability of their speech production. A modified version of Audapter (15, 16) was used to record participants' speech, alter the speech signal when necessary, and play the (potentially altered) signal back to participants. The experiment was conducted in a quiet room with participants seated in front of a computer screen. In each trial, one of the three stimulus words ("bead," "bad," and "bod" in *experiments 1*, *2*, and *4* or "bead," "bad," and "bed" in *experiment 3*) was pseudorandomly selected and displayed on the screen, and participants read it aloud. Speech was recorded at 16 kHz via either a head-mounted microphone (AKG C520, *experiments 1* and *2*) or a desktop microphone (Sennheiser MKE 600, *experiments 3* and *4*). The output of Audapter was played back to participants via closed-back circumaural headphones (Beyerdynamic DT 770) with an unnoticeable delay of ~18 ms, as measured on our system following Ref. 17. All trials were processed through Audapter in the same manner, regardless of whether a perturbation was applied. Speech was played back at a volume of ~80 dB SPL and mixed with speech-shaped noise at ~60 dB SPL, which

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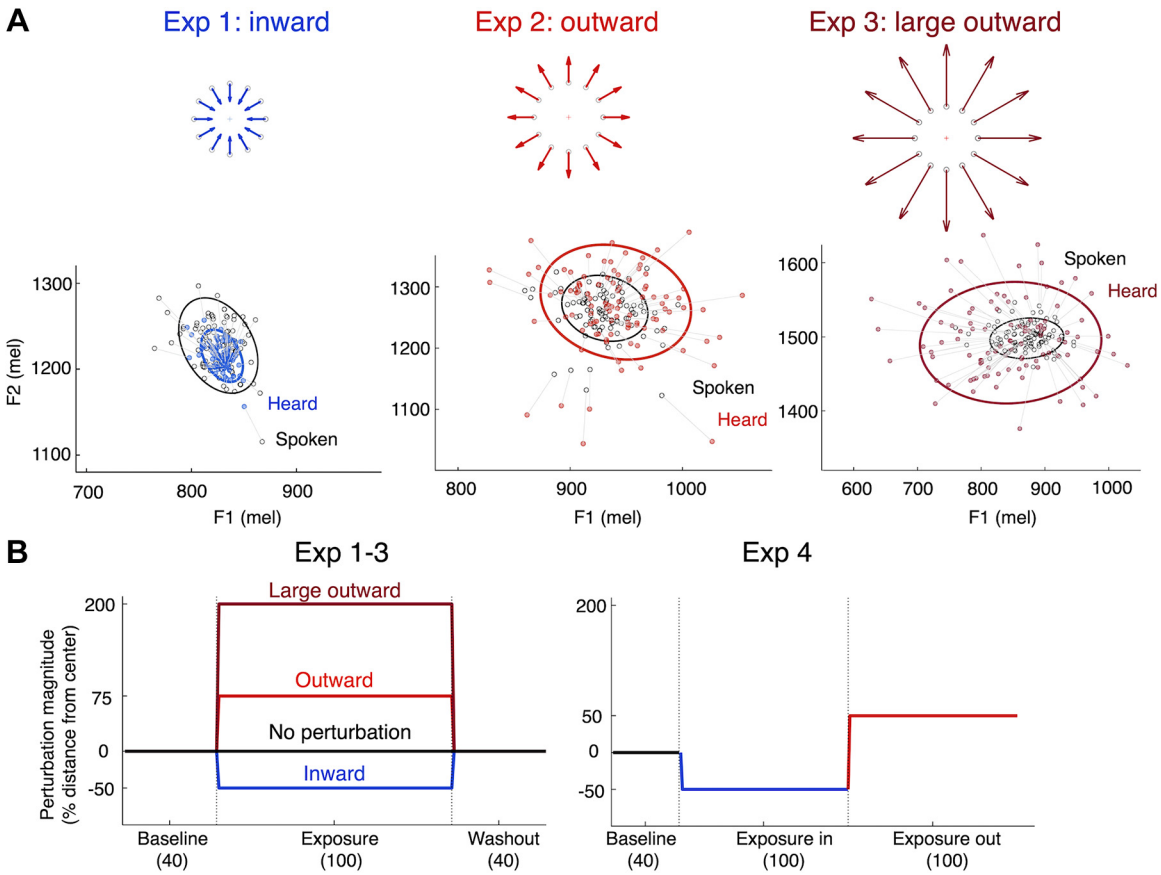

Figure 1. Experiment design. *A*: schematic (*top*) and examples from representative participants (*bottom*) of perturbations applied to speech vowel formants during the exposure phase: inward pushing (*experiment 1*, *left*), outward pushing (*experiment 2*, *center*), and large outward pushing (*experiment 3*, *right*). Note different axis scales in the example data across experiments. Black circles represent the formant values participants produced, and colored circles indicate what was played back to them over headphones. The ellipses represent a 95% confidence interval around the data points of the same color. Note that all formant frequency alterations were applied in mels, a logarithmic measure of frequency. *B*: experimental procedure and magnitudes of the perturbation applied in *experiments 1–3* (*left*) and *experiment 4* (*right*).

served to mask potential perception of the participants' own unaltered speech through air and bone conduction. The volume of speech playback varied dynamically with the amplitude of participants' produced speech.

Experiment-Specific Auditory Perturbation

We designed a modified version of Audapter (18) that is able to affect the perceived variability of speech production by specifying formant perturbations as a function of the current values of F1 and F2 (Fig. 1*A*). It should be noted that all formant frequency alterations were applied in mels, a logarithmic transformation of a signal's frequency such that equal differences in mels are judged by listeners to correspond to equal changes in pitch (19). The transformation from hertz to mel used was mel = 1,127.01048 × log(1 + Hz/700). A participant-specific perturbation field was calculated for each vowel such that every production during the exposure phase was shifted toward (inward-pushing perturbation) or away from (outward-pushing perturbation) the median F1/F2 values of that participant's distribution for that vowel (the vowel "targets"). The magnitude of the perturbation was defined as a percentage of the distance between the currently produced vowel formants and the vowel targets. By scaling the error between the vowel formants and their targets, the inward- and outward-pushing perturbations reduce and magnify, respectively, the perceived variability of speech production.

In *experiment 1* (*inward-pushing perturbation*), participants received a perturbation that shifted every production toward the vowel target. The perturbation was 50% of the distance, in F1/F2 space, between the current formant values and the vowel targets (see Supplemental Fig. S1; all Supplemental Materials are available at https://www.doi.org/10.17605/OSF.IO/STJC9).

In *experiment 2* (*outward-pushing perturbation*), participants received the opposite perturbation, a shift of every production away from these targets, with a slightly larger perturbation magnitude (i.e., 75% of the distance to the vowel target; Supplemental Fig. S1).

In *experiment 3* (*large outward-pushing perturbation*), we aimed to test the possibility that the failure to find the hypothesized reduction in variability in response to the outward-pushing perturbation applied in *experiment 2* was due to inadequate perturbation magnitude. We suspected that a larger perturbation magnitude might be needed to drive participants to produce compensatory reductions in variability. The perturbation magnitude was therefore increased to

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200% of the distance to the vowel target in *experiment 3* (Supplemental Fig. S1), and one stimulus word was changed from a corner vowel word ("bad") to a noncorner vowel word ("bed") to test the effect of the perturbation on noncorner vowels.

In *experiment 4* (*inward-outward pushing perturbation*), we further examined whether limits on articulatory precision prevented participants from reducing their produced variability in response to outward-pushing perturbations in *experiments 2* and *3*: that is, if individuals already produce vowels at the lower limit of variability, they may not be able to produce further variability decreases. In *experiment 4*, participants experienced two exposure phases: first, to an inward-pushing perturbation, which served to increase participants' produced variability above baseline levels, and then to an outward-pushing perturbation. The perturbation magnitudes were 50% of the distance to the vowel target for both inward-pushing and outward-pushing phases.

To control for potential changes in variability over the course of ~500 trials of single-word production, we additionally analyzed an existing data set (18) with a similar experimental structure, though with no auditory perturbation applied (auditory feedback was processed through Audapter in the same manner as the baseline phases in *experiments 1–4*). This control experiment included 460 trials of single-word productions (115 repetitions of each stimulus word in *experiments 1*, *2*, and *4*, "bead," "bad," and "bod," as well as the word "booed," which did not occur in *experiments 1–4* and was not analyzed). To match the experimental design of the present study, the 460 trials were divided into four phases: baseline (30 trials per stimulus), early exposure (30 trials per stimulus), late exposure (30 trials per stimulus), and washout (25 trials per stimulus). The experimental setup for the control data set, including recording, processing, and headphone presentation of speech, was identical to *experiments 1–4*.

Procedure

In all experiments, stimulus words were presented on the computer screen for 1.5 s, one at a time. The interstimulus interval was randomly jittered between 0.25 and 1 s. Participants were instructed to read each word out loud as it appeared.

Each experiment had three phases (Fig. 1*B*). *Experiments 1–3* were divided into baseline, exposure, and washout phases. In the baseline phase (40 trials per stimulus), participants received unaltered auditory feedback and we measured participants' median F1/F2 values for each vowel. These values were subsequently used to calculate the participant-specific perturbation field (see above). The exposure phase followed the baseline phase. In the exposure phase, participants produced each stimulus 100 times while receiving the inward-pushing, outward-pushing, or large outward-pushing perturbation (see experiment-specific auditory perturbation above). *Experiments 1–3* ended with a washout phase in which participants produced each word 40 times with unaltered auditory feedback. In *experiment 4*, after the baseline phase, participants experienced two sequential 300-trial (each stimulus 100 times) exposure phases, first with an inward-pushing perturbation and then with an outward-pushing perturbation. A short self-timed break was given every 30 trials in all experiments.

After they completed the experiment, participants in all four experiments were given a brief questionnaire to assess their awareness of the perturbation as well as whether they adopted any strategy and, if so, what that strategy was.

Quantification and Statistical Analysis

F1 and F2 of produced words were tracked offline with wave_viewer (20), which provides a MATLAB GUI interface to formant tracking using Praat (21). Linear predictive coding (LPC) order and preemphasis values were adjusted individually for each participant. All trials were first checked manually for errors in production (e.g., if the participant said the wrong word). Vowel onset and offset were detected automatically with a participant-specific amplitude threshold, and errors in the location of these automatically defined landmarks were manually corrected with the audio waveform and spectrogram. Vowel onset was marked at the point where periodicity was visible in the waveform and formants were visible in the spectrogram. Vowel offset was marked at the point where formants, particularly F2 and higher, were no longer visible. Reaction time was also calculated for each trial, measured as the time from the start of the recording for that trial (immediately before the stimulus word was presented visually) to the onset of the vowel. Errors in formant tracking were manually corrected by adjusting the LPC order or preemphasis value on a trial-specific basis. In total, 1.6% of the data were excluded because of production errors or unresolvable errors in formant tracking (1.9% in *experiment 1*; 2.2% in *experiment 2*; 1.5% in *experiment 3*; 1% in *experiment 4*). To test whether error rate (i.e., production or unresolvable formant tracking errors) and reaction time differ between experiments and across experimental phases, mixed ANOVAs were conducted separately for the error rate and reaction time, with phase as a within-subject factor and experiment as a between-subject factor. For each trial, F1 and F2 values were calculated by averaging formants from a 50-ms segment at both the beginning (vowel onset) and the middle (vowel midpoint) of each vowel.

The primary goal of the analysis was to test how variability changed across the different phases of each experiment. For offline analysis, the exposure phase was equally divided into early exposure and late exposure phases to make sure each phase contained a similar number of trials. Variability within each experimental phase was calculated as the average of the two-dimensional (2-D) distances in F1/F2 space between each production of a vowel and the center of the distribution for that vowel in that phase, measured from the first 50 ms of vowel. To test how variability may change in specific dimensions, we additionally calculated formant variability separately along the F1 and F2 axes, as well as along the major and minor axes of produced variability in the baseline phase. The variability along the F1 and F2 axes was defined as the standard deviation (SD) of F1 and F2 values of all productions of a stimulus word during each phase. For the major and minor axes of variability, an ellipse that represents a 95% confidence

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interval of trials in F1-F2 spaces was fitted for each stimulus word and each experimental phase with the principal components method. The vector representing the F1 and F2 values for each trial was projected into a component along the major axis of the fitted ellipse and a component along the minor axis perpendicular to the major axis. The variability along the major and minor axes was defined as the standard deviation (SD) of projected values along that axis of all productions of a stimulus word during each phase.

We additionally measured vowel centering, a measure of within-trial correction for variability, calculated by the change in variability from vowel onset (first 50 ms) to vowel midpoint (middle 50 ms). In other words, vowel centering is the difference in 2-D distances, measured from the first and middle 50 ms of vowel. Vowel centering allows us to determine whether participants altered their within-trial control of variability in response to the perturbation (22–24). Centering was measured separately for each vowel in each experimental phase.

Repeated-measures analyses of variance (ANOVAs) were conducted separately for the variability and centering results and for each experiment, with phase and vowel identity as within-subject factors. For the control data and *experiments 1–3*, data from baseline, late exposure, and washout phases were included in the repeated ANOVAs, whereas data from baseline, inward-pushing exposure, and outward-pushing exposure phases were included in *experiment 4*. We assumed that any production changes (variability or centering) induced by auditory perturbations reached a relatively steady state during the late exposure phase. Post hoc comparisons (paired *t* tests) were only conducted in the event of a significant main effect of phase or interaction. As an additional measure of the response to the auditory perturbations, baseline-normalized changes in variability during late exposure in each experiment were compared to control data with independent *t* tests. As an exploratory analysis, multiple regression was conducted in each experiment to determine whether produced variability changes could be predicted by baseline variability and vowel identity (random intercepts were included for participant). Finally, variability changes along F1/F2 or major/minor axes were examined separately by three-way repeated-measures ANOVAs that included phase, vowel identity, and axis (i.e., F1/F2 or major/minor) as within-subject factors. The significance level for all statistical tests was $P < 0.05$, with a Bonferroni correction for multiple comparisons for post hoc tests.

All statistical analyses were conducted in R (25). Repeated-measures ANOVAs and *t* tests were conducted with the rstatix package (26), in which partial eta squared (η_p^2) and Cohen's *d* were calculated for repeated ANOVAs and *t* tests, respectively, to determine effect size for statistically significant effects. Greenhouse–Geisser correction was applied automatically to correct the degrees of freedom when sphericity was violated. Multiple regression models were constructed with the package lme4 (27). Supplemental materials, data, and associated code are available at https://www.doi.org/10.17605/OSF.IO/STJC9. Some functions rely on additional code available at https://github.com/carrien/free-speech.

Model Simulations

To assess the potential mechanisms underlying the patterns of variability changes observed in *experiments 1–4*, we conducted a simulation of speech behavior using a version of the well-established state-space models that have been used in studies of sensorimotor adaptation to sensory perturbations in both limb (28–30) and speech (31, 32) motor control. Importantly, analogous models have also been used successfully in studies of sensorimotor corrections for self-produced variability in reaching (33–35), similar to the present experiments. The model is

$$m_t = Am_{t-1} + Be_{t-1}$$
$$x_t = m_t + N(0, K)$$
$$e_t = \pm Gx_t$$

This model assumes that the intended production (m) on a given trial (t) aims to achieve a particular target (by convention set to 0) based on a weighted contribution of the production on the previous trial ($t - 1$) and the error (e) experienced on the previous trial. A is the forgetting factor that determines the contribution of the previous trial, and B represents the sensitivity of the system to errors. The error e is the difference between the actual production outcome (x) and the target, where the production x is the result of the intended production m plus noise (N) drawn from a Gaussian distribution with a mean of 0 and a standard deviation of K. To account for the gains applied to the auditory feedback in this experiment, the observed outcome x is multiplied by a gain factor G to derive the error. Aside from the addition of the gain factor G, this is identical to the formulation in Ref. 33, if $A = 1$.

Although the state-space model is often used to model sensorimotor adaptation to external perturbations, the addition of the noise term on the final motor output also permits modeling of correction for self-produced variability (33–35). For simplicity, we initially set A to 1 (33) and B to 0.1 given previous results fitting state-space models to adaptation in speech (31, 32). We then estimated an initial value of K that would generate an observed distribution similar to the experimentally observed variability in the baseline phase across experiments (roughly 30 mels). Because the errors in this model are generated stochastically, we need a large number of simulations to achieve an accurate estimate of the underlying distribution of variability that would be generated through this model. To that end, we ran batches of 1,000 simulations while varying K. Each simulation consisted of 40 trials, equivalent to the number of trials used to estimate variability in our experimental data. Our initial simulations indicated that setting K to 30 resulted in a mean observed variability close to the observed values in the baseline phase across experiments. Varying A within the range reported in Ref. 31 had very minor effects on the results; the effects of varying B are explored below.

Our goal in modeling was to assess the potential causes of the increase in variability observed in the behavioral data in *experiments 1–4*. To do this, we systematically varied the underlying variability K as well as the sensitivity to errors B. K varied from 30 to 40 in steps of 0.5. B varied from 0 to 1 in steps of 0.05, where 0 would represent no correction for observed errors and 1 would represent full correction. For

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each step of K and B, 1,000 simulations (40 trials each) were conducted. The standard deviation of x was calculated for each simulation, and the mean of these standard deviations was calculated to generate an estimate of the expected variability with that particular parameter set. We ran separate simulations for the inward-pushing perturbation in *experiment 1* (setting G to 0.5) and the outward-pushing perturbation in *experiment 2* (setting G to 1.75).

RESULTS

Overall Variability Changes

Over four experiments, we implemented an auditory perturbation designed to increase or decrease participants' perceived trial-to-trial variability without affecting their overall mean. To confirm that this was achieved, the perturbations applied to F1 or F2 frequencies during the exposure phase in each experiment were averaged for each participant and compared against 0 (no mean formant value change) with one-sample t tests. The mean F1 perturbations ranged between 2.5 and 13.4 mels in *experiments 1–4* and significantly differed from 0 only in *experiment 3* [large outward pushing; mean F1 perturbation: 13.4 mels, $t(20) = 2.47$, $P = 0.023$]. Similarly, the mean F2 perturbations ranged between −1.23 and 12.39 mels in *experiments 1–4*, with no significant difference from 0 in any case (uncorrected $P > 0.05$). It is worth mentioning that the just noticeable difference (JND) in F1 and F2 for isolated English vowels is around 14 mels and 20 mels, respectively (36).

We then measured how participants changed their produced variability after the perceived variability had been increased or decreased. As a control, we analyzed an existing data set (18) with no auditory perturbation. As expected, the control group showed no change in variability over the course of the experiment [Fig. 2*A*; $F(2,48) = 0.434$, $P = 0.650$].

Participants exposed to the inward-pushing perturbation (*experiment 1*) increased produced variability [Fig. 2*B*; main effect of phase: $F(2,46) = 7.55$, $P < 0.001$, $\eta_p^2 = 0.25$] while the perturbation was applied (+5.0 mels, $t = 2.98$, $P = 0.02$, $d = 0.609$) as well as after it was removed (+4.4 mels, $t = 3.32$, $P = 0.009$, $d = 0.677$). The baseline-normalized variability was significantly larger in *experiment 1* than in the control data during the exposure phase ($t = -2.91$, $P = 0.006$, $d = 0.84$). Unexpectedly, participants exposed to the outward-pushing perturbation (*experiment 2*) also increased produced variability [Fig. 2*C*; main effect of phase: $F(2,40) = 7.93$, $P < 0.001$, $\eta_p^2 = 0.28$]. Again, the increase in response to the outward-pushing perturbation led to a significant difference between *experiment 2* and control data in baseline-normalized variability during the exposure phase ($t = -3.19$, $P = 0.003$, $d = 0.94$). However, unlike the maintained variability change seen in the inward-pushing perturbation, the increased variability during exposure (+4.3 mels, $t = 3.53$, $P = 0.006$, $d = 0.75$) returned to near-baseline levels when the outward-pushing perturbation was removed (+1.6 mels, $t = 1.57$, $P = 0.393$).

We reasoned that the failure to find the hypothesized reduction in variability in response to the outward-pushing field could potentially be attributed to the size of the perturbation used. Although this perturbation was slightly larger than in *experiment 1* (75% vs. 50% of the distance to vowel target), it may still have been too small to induce participants to reduce their variability. *Experiment 3* (large outward-pushing perturbation) aimed to delineate this by testing another group of participants who received outward-pushing perturbations with a 200% increase in distance to the center of vowel distribution (vs. 75% in *experiment 2*). However, participants exposed to this large outward-pushing perturbation did not change their produced variability [Fig. 2*D*; main effect of phase: $F(1.55,30.95) = 1.39$, $P = 0.26$]. No significant difference in normalized variability was observed between *experiment 3* and control data during the exposure phase ($t = -1.49$, $P = 0.144$). This result also rules out the possibility that the increase in variability observed in *experiments 1* and *2* is a general, nonspecific consequence of the auditory perturbation itself (that is, that any kind of auditory perturbation leads to an increase in formant variability).

Together, the results of both experiments employing outward-pushing perturbation fields (*experiments 2* and *3*) suggest that these perturbations do not drive participants to produce compensatory reductions in variability. One possibility for this behavior is that speech movements are already produced at or near the lower limit of an individual speaker's precision ability. *Experiment 4* (inward-outward pushing perturbation) aimed to test this possibility by examining whether an outward-pushing perturbation can reduce produced variability of participants after their variability has increased above baseline levels because of exposure to an inward-pushing perturbation as seen in *experiment 1*. Participants significantly changed their produced variability during the course of *experiment 4*, reflected by a main effect of phase [Fig. 2*E*; $F(2,38) = 9.71$, $P < 0.001$, $\eta_p^2 = 0.34$]. As expected given the results of *experiment 1*, variability increased when the inward-pushing perturbation was applied [4.1 mels, $t = 4.0$, $P = 0.002$, $d = 0.89$; comparison with control data: $t(39.7) = -2.56$, $P = 0.014$, $d = 0.77$]. However, participants did not change their produced variability back to the baseline when receiving the outward-pushing perturbation in the following phase [+4.1 mels, $t = 4.04$, $P = 0.002$, $d = 0.90$; comparison with control data: $t(41.6) = -2.23$, $P = 0.031$, $d = 0.67$]. While this result replicates the increase in variability observed during the exposure phase in *experiments 1* and *2*, it suggests that the failure to find the expected reduction in variability in *experiments 2* and *3* was not caused solely by a "lower limit" on variability.

Analysis of the error rate (mixed ANOVAs) confirmed that the error rate did not significantly differ between experiments [main effect of experiment: $F(2,64) = 1.49$, $P = 0.233$], nor did it change across phases [main effect of phase: $F(1.3,85.5) = 2.0$, $P = 0.155$; interaction between experiment and phase: $F(2.7,85.5) = 2.1$, $P = 0.116$]. Although reaction times did increase over the course of each experiment [main effect of phase: $F(1.1,67.5) = 10.78$, $p = 0.001$, $\eta_p^2 = 0.14$], this change did not differ between experiments [interaction between experiment and phase: $F(2.1,67.5) = 2.01$, $P = 0.139$; main effect of experiment: $F(2,64) = 0.73$, $P = 0.484$].

Finally, although it was not the primary focus of the study, we evaluated whether participants adjusted their variability differently along the major/minor axes of variation using three-way repeated ANOVAs including three within-subject factors (phase, vowel, axis). This analysis replicated the

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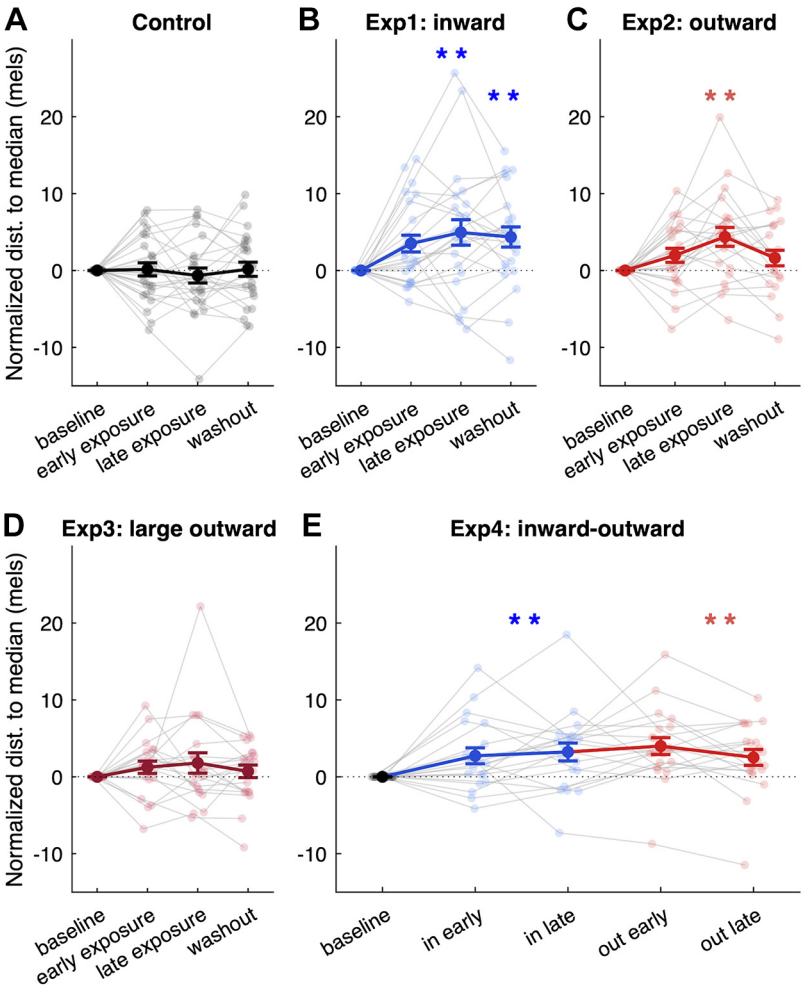

Figure 2. Baseline-normalized variability changes. Individual (small transparent dots, thin lines) and group mean (large solid dots, thick lines) baseline-normalized variability (normalized by subtracting the average value in the baseline from the remaining trials) in the baseline, exposure, and washout phases in control (*A*) and *experiments 1–4* (*B–E*). Error bars show SE. **Significant change ($P < 0.001$) from baseline.

results of overall variability changes (distances in F1-F2 spaces): a significant main effect of phase was found in *experiments 1*, *2*, and *4* but not in the control data or in *experiment 3*. Perhaps more interestingly, no significant two-way interaction between phase and measure was found in any of these experiments, suggesting that participants adjusted their variability along the major and minor axes similarly. Similar results were found from models of F1 and F2 variability. See Supplemental Figs. S2 and S3 and Supplemental Tables S1 and S2 for detailed results and statistics.

Centering Changes

To determine whether participants adjusted their within-trial control of variability in response to the perturbation, we additionally measured vowel centering (22–24), the reduction in variability from vowel onset (first 50 ms) to vowel midpoint (middle 50 ms). Similar to the analyses on variability, the control group did not show any change in centering over the course of the experiment [Fig. 3*A*; $F(2,48) = 1.45$, $P = 0.244$]. However, unlike the overall variability changes observed above, no change in centering was seen in participants who received the inward-pushing perturbation [*experiment 1*, Fig. 3*B*; $F(1.49,34.27) = 0.06$, $P = 0.896$; comparison with control data: $t = -1.56$, $P = 0.306$]. In contrast, centering did increase when participants were exposed to the outward-pushing perturbation [*experiment 2*, Fig. 3*C*; main effect of phase: $F(2,40) = 3.94$, $P = 0.027$, $\eta_p^2 = 0.165$], suggesting that these participants became more responsive to errors. This increase in centering during exposure [+2.2 mels, $t = 2.68$, $P = 0.042$, $d = 0.57$; comparison with control data: $t(43.6) = -3.4$, $P = 0.001$, $d = 0.99$] was not retained during the washout phase (+0.1 mels, $t = 0.09$, $P = 1.000$). Contrary to the increase in centering observed in *experiment 2*, participants who received a large outward-pushing perturbation in *experiment 3* (i.e., 200% increase in distance to the vowel target) did not show an increase in centering over the course of the experiment [Fig. 3*D*; $F(2,40) = 0.87$, $P = 0.428$], actually tending to slightly decrease centering both during exposure [comparison with control data: $t(33.5) = -0.2$, $P = 0.843$] and in the washout phase. In *experiment 4* (inward-outward pushing perturbation), participants showed changes in centering over the course of the experiment, indicated by a marginally significant main effect of phase [Fig. 3*E*; $F(1.4,26.5) = 3.15$, $P = 0.075$, $\eta_p^2 = 0.142$]. More specifically, the results replicated the patterns observed in *experiments 1* and *2*: the initial inward-pushing perturbation did not induce any significant increase in centering (+0.53 mels, $t = 0.32$, $P = 1.000$; comparison with control data: $t = -1.78$, $P = 0.087$), whereas the subsequent outward-pushing perturbation did (+ 2.7 mels, $t = 1.92$, $P = 0.71$, $d = 0.43$; comparison with control data: $t = -2.47$, $P = 0.02$, $d = 0.79$).

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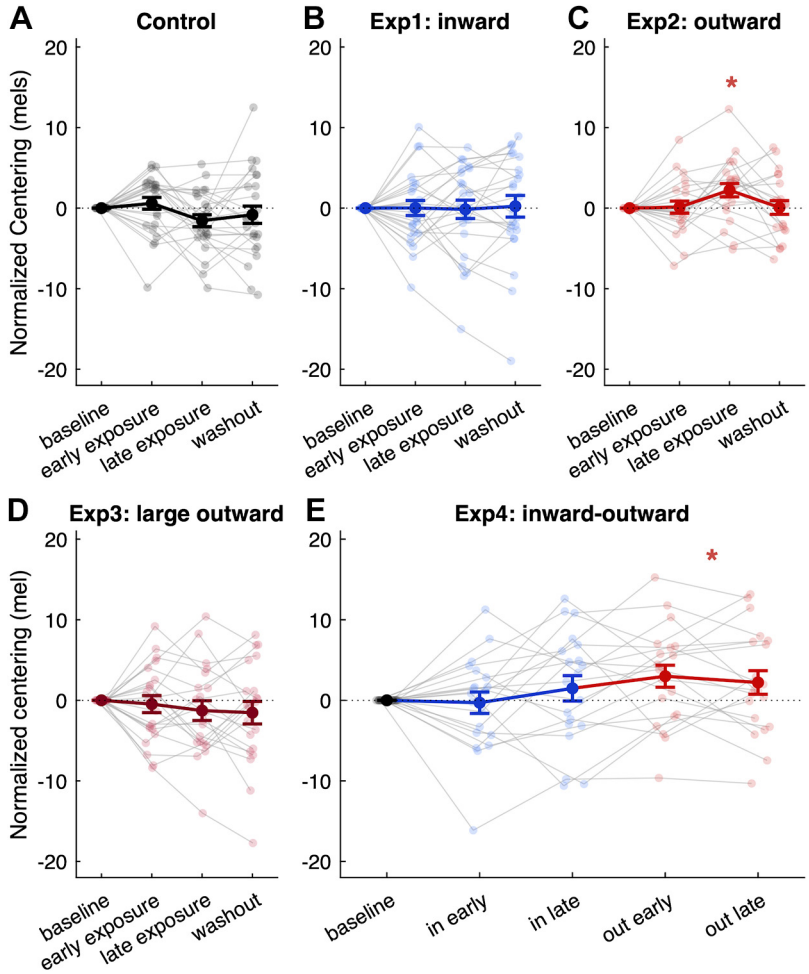

Figure 3. Baseline-normalized centering changes. Individual (small transparent dots, thin lines) and group mean (large solid dots, thick lines) baseline-normalized centering (normalized by subtracting the average value in the baseline from the remaining trials) in the baseline, exposure, and washout phases in control (*A*) and *experiments 1–4* (*B–E*). Error bars show SE. *Significant change ($P < 0.05$) from baseline.

Correlation between Variability Change and Baseline Variability

Multiple regression was conducted in each experiment to determine whether changes in produced variability could be predicted by baseline variability and vowel identity at the individual level (see Fig. 4). Results showed that baseline variability was not predictive of the individual change in variability observed during the late exposure phase in either the control ($\beta = -0.20$, $P = 0.065$) or *experiment 2* (outward-pushing perturbation, $\beta = -0.06$, $P = 0.646$). However, this relationship was observed in *experiment 1* (inward-pushing perturbation, $\beta = -0.52$, $P < 0.001$), such that participants with lower variability in the baseline phase showed larger variability increases. In *experiment 3* (large outward-pushing perturbation), where there was no consistent change in variability over the course of the experiment, baseline variability was nonetheless predictive of individual changes in variability ($\beta = -0.50$, $P < 0.001$): participants with higher variability in the baseline phase tended to decrease variability, and vice versa. *Experiment 4* (inward-outward pushing perturbation) replicated the result from *experiment 1*: a significant correlation was observed between baseline variability and variability change induced by inward-pushing perturbation ($\beta = -0.29$, $P = 0.015$). Perhaps surprisingly given the results of *experiment 2*, a similar relationship between baseline variability and variability change was also observed during later outward-pushing perturbation ($\beta = -0.37$, $P < 0.001$). However, it should be noted that there was a highly significant correlation in variability between the two perturbation phases ($\beta = 0.54$, $P < 0.001$), suggesting that the correlation observed during the outward-pushing perturbation is likely a carryover effect of the inward-pushing perturbation that happened between baseline and outward-pushing phases.

State-Space Simulations

Our behavioral experiments showed the somewhat counterintuitive result that produced variability increased in response to auditory perturbations whether they decreased or increased perceived variability. We hypothesized that a superficially similar response may actually arise from different mechanisms in the two perturbation paradigms. Specifically, the inward-pushing perturbation may have led to increases in controlled variability as participants relaxed their constraints on movement outcomes; conversely, the outward-pushing perturbation may have increased participants' sensitivity to errors (consistent with the observed increase in centering), potentially leading to an increase in produced variability without any direct changes to variability constraints. We tested this hypothesis with a modified version of the typical state-space model that has been

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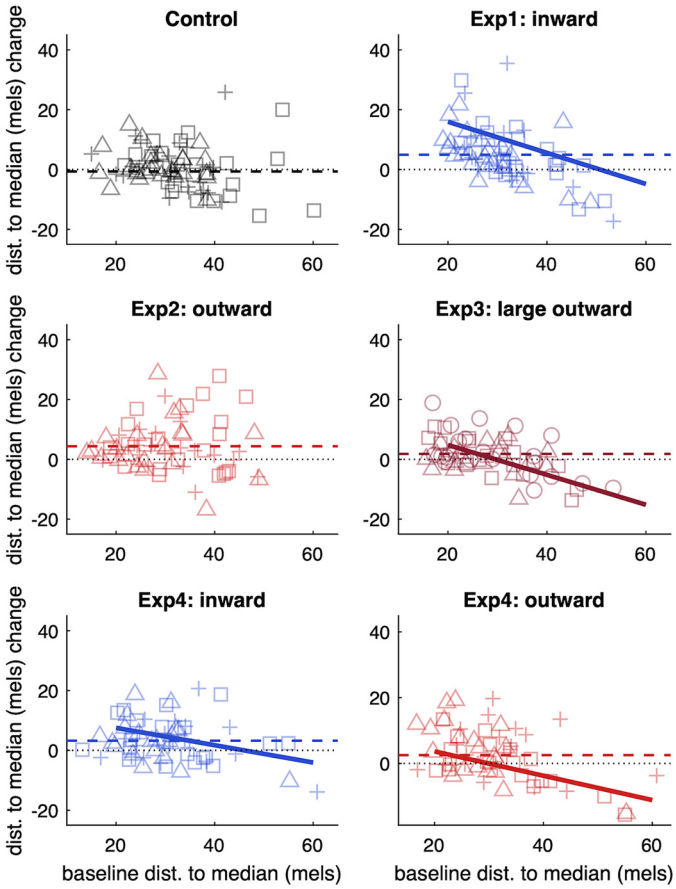

Figure 4. Correlation between average baseline variability and variability change across individuals. Each data point represents the average production of 1 stimulus word, indicated by different markers. Each participant thus contributed 3 data points. Note that the stimulus words were "bead," "bad," and "bed" in *experiment 3* and "bead," "bad," and "bod" in the other experiments. Regression lines with significant regression coefficients (β) of baseline variability are indicated by solid lines. The group-averaged (baseline normalized) variability changes are indicated by colored dashed lines.

shown to accurately account for trial-to-trial corrections for self-produced variability in reaching (33–35) by systematically varying the magnitude of controlled motor variability and the sensitivity of the system to observed errors (K and B, respectively; see METHODS for details). Results of these simulations are shown in Fig. 5. We found that, predictably, increases in the underlying motor variability K increased the observed variability in motor production for both the inward- and outward-pushing perturbations (Fig. 5*C*). Values of K near 35 produced observed variability measures consistent with those in both *experiments 1* and *2* (Fig. 5*D*). Conversely, we found that changes in error sensitivity had differential effects on motor variability depending on the perturbation field. For the inward-pushing perturbation, variation in the error sensitivity B had very minor effects on motor output. Conversely, for the outward-pushing perturbation, increases in error sensitivity led to large increases in motor variability (Fig. 5*E*). Values of B near 0.3 resulted in a good match for the observed variability in *experiment 2*, whereas no values of B provided a good match for *experiment 1* (Fig. 5*F*). In the latter case, the maximum increase in observed variability over baseline was 3.7 where $B = 1$, still substantially less than the experimentally observed value. These data suggest that changes in error sensitivity may explain the pattern of behavior only in the outward-pushing condition whereas changes in underlying motor variability could potentially provide explanations for both conditions (see also Supplemental Fig. S4).

To disentangle the two potential sources of variability change in the outward-pushing condition, we further examined whether changes in these parameters could explain the pattern of behavior not only in the exposure phase, when the gain on error was experimentally altered ($G = 0.5$ or 1.75), but also in the washout phase, after the perturbation is removed ($G = 1$). Results can be seen in Fig. 5*G*, with a summary of corresponding participant behavior observed in inward-pushing and outward-pushing conditions in Fig. 5*H*. The assumption in these simulations is that any changes in parameters K and B induced in the exposure phase are retained short term in the washout phase. All values are chosen to match behavior in the exposure phase based on simulations shown in Supplemental Fig. S4. Increasing the underlying variability K from 0 to ~35 provides a qualitative match to the behavior observed in the inward-pushing condition (blue lines in Fig. 5, *G* and *H*), with increases in variability observed in both the exposure and washout phases. Increasing K to ~34 in the outward-pushing condition (dashed red line in Fig. 5*G*) predicts the observed increase in variability in the exposure phase but incorrectly predicts that this increase is retained in the washout phase. Conversely, increasing error sensitivity B from 0.1 to ~0.3 provides a better match for the observed data in this case (solid red lines in Fig. 5, *G* and *H*), with an increase in variability that peaks in the exposure phase before decreasing to slightly above the baseline value in the washout phase. These simulations suggest that changes in error sensitivity are a more likely explanation of the behavior in the outward-pushing condition than changes in (only) underlying motor variability.

The state-space model captures individuals' "one-shot" adaptation behavior, i.e., compensatory changes in the trial following a sensory error (37). It is therefore possible that the increased variability observed in *experiment 2* (outward-pushing perturbation) was driven by attempts to compensate for perceived errors in previous trials rather than any change to control parameters. In the model, the perceived outcome is multiplied by a gain factor G to derive the perceived trial-to-trial error (G was set to 0.5 for the inward-pushing perturbation, which decreases perceived variability, and to 1.75 for the outward-pushing perturbation, which increases perceived variability). This perceived error term is then multiplied by the error sensitivity B to drive trial-to-trial corrections in motor behavior. As shown in Fig. 5*E*, when B was fixed at 0.1 (estimated based on previous results fitting state-space models to adaptation in speech), changing G to simulate the auditory perturbations had very minor effects on the observed variability. To match the increase in magnitude observed in response to the outward-pushing perturbation in *experiment 2* by changing G alone, the baseline value for B would need to be roughly 0.5 or higher. We believe that such a high intrinsic gain on errors is unlikely:

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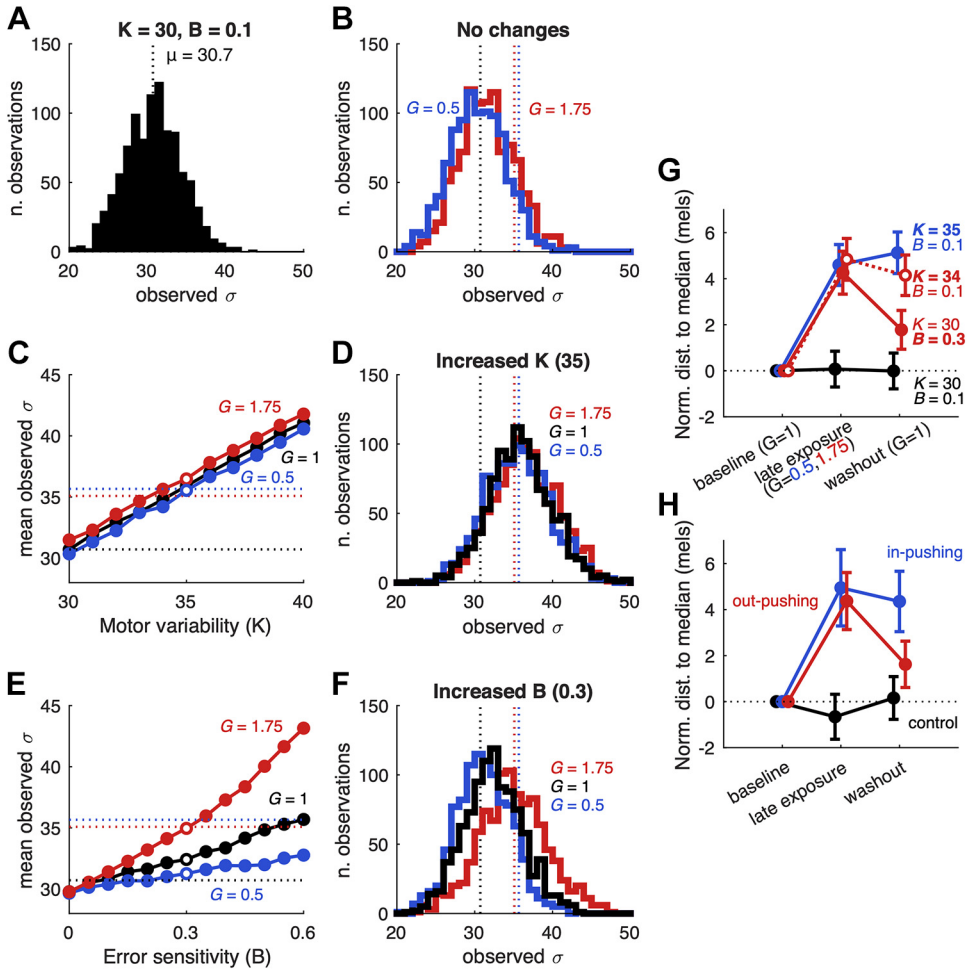

Figure 5. State-space simulations of motor variability. For all panels, the dashed black line represents the mean variability (μ) with model parameters set to match participant behavior in the baseline phase, the dashed blue line represents the increase in variability observed in *experiment 1* (4.9 mels above baseline), and the dashed red line represents the increase in variability observed in *experiment 2* (4.4 mels above baseline). *A* shows distributions of observed variability (σ) for 1,000 simulations where the gain applied to the observed error is 1, simulating the baseline phase in both *experiments 1* and *2*. *B* shows similar distributions from simulations with gains set to 0.5 (blue) and 1.75 (red), mirroring the perturbations applied in *experiment 1* and *experiment 2*, respectively, with no changes to other model parameters. *C* shows the result of systematically varying underlying motor variability *K* on observed variability with gains set to 1 (black), 0.5 (blue), and 1.75 (red). The open circles in *C* represent the value for *K* used in the simulations shown in *D*. *D* shows the distribution of observed variability with *K* set to 35, chosen to roughly match the increase in variability observed in *experiments 1* and *2*. *E*: as for *C*, showing the effect of varying the error sensitivity parameter *B*. Values of $B > 0.6$ (not shown) resulted in observed variability values >50 where $G = 1.75$ and <34 where $G = 0.5$. *F* shows the distribution observed with *B* set to 0.3, chosen to match the increase in variability observed in *experiment 2*. *G* shows simulations where *K* or *B* is increased in both the exposure and washout phases. *H* shows experimental data as a comparison with modeling in *G*. In the simulations, the error gain *G* in each phase is set to match the experimental value. Compared with the baseline, increasing *K* to ~35 produces increases in variability in both the exposure and washout phases in the inward-pushing condition, matching experimental data (blue). Similarly increasing *K* to ~34 in the outward-pushing condition produced similar results, inconsistent with experimental data (dashed red line). Conversely, increasing *B* to ~0.3 in this condition (solid red line) produces a peak in variability in the exposure phase followed by a drop that is still above baseline in the washout phase, matching experimental data.

the existing literature fitting similar models to human behavior in both speech and reaching consistently shows that feedback gains are <0.2 (28, 29, 31, 32). Thus, we believe that the observed increases in produced variability are unlikely to be caused by the perturbation alone (i.e., the gain factor *G*) without any changes to variability constraints (*K*) or error sensitivity (*B*). Although it is not possible to directly fit these state-space equations to our present data because of the insufficient number of trials, excluded trials due to poor formant tracking, and multiple interleaved targets, these simulation results nonetheless indicate that the observed increase in *experiment 1* can only be explained by an increase in the underlying motor variability, whereas the increase observed in *experiment 2* could arise through either an increase in controlled variability or an increase in the sensitivity to auditory errors.

Awareness of Perturbation

After the experiment, participants were informed that their speech might have been manipulated and were asked if they thought they were in a group with true feedback or one with some manipulated feedback, as well as what they thought the manipulation was. Across *experiments 1*, *2*, and *4*, only one participant (out of 66 total) correctly identified the perturbation as a change to their vowels. Although an additional 24 participants indicated that they thought they

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were in the group with manipulated feedback after our prompt, they reported a range of other perceived changes: a manipulation of the pitch/tone (6 participants), voice volume (3 participants), speech rate/duration (3 participants), background noise (4 participants), or other nonspecific distortions to speech (e.g., "sounds weird," "just guessing," 8 participants). In contrast, nearly all participants (18/21) in *experiment 3* (large outward pushing) reported that they thought they were in the group with manipulated feedback and almost half (9/21) correctly identified it as a manipulation to their vowels. No participants reported a strategy that aimed to address the applied perturbation in any of the four experiments.

DISCUSSION

In a set of four experiments, we examined whether variability can be actively regulated in a complex, well-practiced motor task: speech production. Specifically, we examined whether participants would adjust their produced variability when they were exposed to real-time auditory perturbations that increased or decreased their perceived variability. Our results showed that introducing a perturbation that reduces perceived variability (*experiment* 1: inward pushing) leads to increases in produced vowel variability that remain even when normal feedback is restored, suggesting that variability is monitored and regulated over relatively long timescales. Perhaps surprisingly, a perturbation that increases perceived variability (*experiment 2*: outward pushing) also increased produced variability, though such variability change was not maintained when the perturbation was removed. In *experiment 3*, participants who received a large outward-pushing perturbation (i.e., a 200% increase in distance to the vowel target) did not change their produced variability over the course of the experiment. This rules out the possibility that the failure to find the hypothesized reduction in variability in response to the outward-pushing perturbation (*experiment 2*) is due to inadequate perturbation magnitude. This result also suggests that the variability increases observed in both *experiments 1* and 2 are unlikely to be caused by a nonspecific response to the presence of any auditory perturbation. Finally, we found that an outward-pushing perturbation cannot reduce participants' produced variability even after their variability has increased above baseline levels as a result of the inward-pushing perturbation (*experiment 4*: inward-outward pushing).

To our knowledge, this is the first study showing that variability in speech production can be modulated, consistent with recent theories that have highlighted the adaptive value of motor variability in other motor domains (38–40). From this perspective, motor variability can be actively generated, regulated, and used by the brain to improve motor performance, reduce costs, and explore new solutions (41). Early evidence shows that skilled performers are able to upregulate the level of motor variability in each joint of the upper arm to meet the change of task demands/constraints, whereas less skilled performers, in comparison, tend to have rigidly fixed motor variability that is not fine-tuned with task constraints (Ref. 42; see Ref. 43 for a review). More recent work using computational models also found that force variability and the resulting kinematic variability are not generated primarily by random motor noise and emphasize the importance of other sources of force variability that can be tuned as needed by distributed sensorimotor systems (44). The results from the present study extend previous work and provide support for this perspective by showing that the motor system closely monitors sensory variability and uses such information to actively regulate the motor variability, even in complex and well-practiced behaviors such as natural speech. Note that this work focuses on the ability of the central nervous system (CNS) to modulate variability in behavior, without directly probing the mechanisms through which this could be accomplished. Previous studies in reaching have shown changes in impedance or cocontraction in the context of different accuracy constraints, suggesting that the CNS may use impedance modulation as a way to regulate motor variability (45, 46). Future work is needed to clarify how the CNS might implement or achieve motor regulation during speech production.

Surprisingly, participants exposed to both inward-pushing and outward-pushing perturbations increased their produced variability. These results could be interpreted as a general variability increase induced by either repetitive productions of many utterances or a nonspecific auditory perturbation (that is, any kind of auditory perturbation would lead to an increase in formant variability). However, the results from our data as well as previous studies indicate that both of these possibilities are unlikely. First, in analyzing an existing data set where participants produced 460 utterances with normal auditory feedback (18), we found that speakers did not significantly change their produced variability. This confirms that under normal circumstances speakers produce vowels with a level of variability that is relatively stable over time. Second, previous work has shown that participants do not change their formant variability in response to a consistent auditory perturbation of both F1 and F2 (i.e., shifts of 240 Hz in F1 and 300 Hz in F2) (47). Results from *experiment 3* are consistent with this result, as participants who received a large outward-pushing perturbation did not exhibit any significant change in their variability. These results, taken together, rule out the possibility of a general nonspecific increase in variability induced by auditory perturbations.

In our experimental data, the increased variability induced by the auditory perturbation was maintained even when normal feedback was restored in *experiment 1*, but not in *experiment 2*. This difference suggests that different mechanisms may have led to the increased variability observed in the two experiments. As an attempt to disentangle these potential mechanisms, we conducted a simulation using a state-space model of error correction. Model simulations identified two distinct mechanisms that could lead to the observed increase in produced variability: an increase in controlled variability and an increase in the sensitivity to auditory errors. More specifically, these simulations indicated that the observed increase in *experiment 1* (inward pushing) can only be explained by an increase in controlled variability, whereas the increase observed in *experiment 2* (outward pushing) could arise through either of these two mechanisms. Importantly for *experiment 2*, additional simulations showed that an increase in error sensitivity, but not

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an increase in variability, reproduces the experimentally observed behavior in both the exposure and washout phases (see Fig. 5). These modeling results suggest that the increase in observed variability in response to the inward-pushing perturbation in *experiment 1* was likely driven by a relaxation of controlled variability, as the perturbation "frees" the motor system to be more variable without any loss in perceived accuracy. Conversely, the results from *experiment 2* are most consistent with an increase in error sensitivity caused by the outward-pushing field rather than a change in controlled variability. Consistent with this difference, we found a significant correlation between produced variability changes and baseline variability in *experiment 1* but not in *experiment 2*. One possible explanation for the pattern observed in *experiment 1* is that participants who are naturally more variable may take advantage of the natural consequences of the inward-pushing perturbation to reduce overall perceived variability, whereas less variable individuals may relax their (presumably stricter) regulation of variability without negatively affecting their perceived variability. In contrast, in *experiment 2* we would not expect that changes in error sensitivity would be related to levels of baseline variability. In brief, the combined results of behavioral and model simulation point to the two distinct mechanisms that may have led to the observed increase in variability in response to inward-pushing and outward-pushing perturbation.

It is worth mentioning that although it would be ideal to compare the predictions generated by the state-space model to empirical estimates of error sensitivity, limitations of the experimental design prevent us from being able to estimate such parameters. Even with recent advances in this regard (34), accurate estimation of these parameters requires 100–200 sequential trials. In all experiments, participants produced 40–50 repetitions of each word per phase, in a pseudorandomized order. Thus, our protocol provides both an insufficient number of trials as well as a potential confound of producing multiple intervening movements between repetitions of the same item. Future work should explore whether current methods that are known to provide accurate estimates of error sensitivity in reaching could be applied to speech motor control with a more appropriate experimental design.

We additionally measured vowel centering, a measure of within-trial correction for variability (22–24). Unlike the overall variability changes observed in both kinds of perturbation, an increase in centering was only seen in participants who received the outward-pushing perturbation in *experiment 2* and in the outward-pushing phase of *experiment 4*. This suggests that participants exposed to outward-pushing perturbations became more responsive to within-trial errors (i.e., larger within-trial feedback gains). Although it is not clear that such an increased sensitivity to errors for within-movement corrections is directly related to the error sensitivity related to cross-trial changes (i.e., gains on trial-to-trial learning), it is possible that error sensitivity is shared across both processes. This would be consistent with the fact that we see increased centering only in *experiment 2*, precisely where modeling results suggest an increase in trial-to-trial error sensitivity.

The only case where we did not see an increase in centering or variability was *experiment 3* (large outward-pushing perturbation). It is possible that the larger perturbations used in this experiment were discounted by the sensorimotor system, such that they had limited effects on speech production. This is consistent with previous studies that have similarly shown attenuated responses to large auditory feedback perturbations (48, 49), potentially because large perturbations are treated as externally induced rather than self-produced errors (50). This is also in line with the perturbation awareness results: almost half of the participants in *experiment 3* correctly recognized the perturbation applied on vowels, whereas only one participant did across all other experiments.

Together, the results of *experiments 2–4* suggest that a sensory perturbation that increases perceived variability does not drive participants to produce compensatory reductions in speech variability, contrary to previous results in nonspeech motor control that showed that task-relevant variability can be reduced when needed or after repeated practice (8, 51, 52). One potential explanation for our failure to observe the hypothesized decrease in variability in response to the outward-pushing perturbation in *experiment 2* is that speech is already produced at the lower limits of possible task-related variability, consistent with the predictions of the uncontrolled manifold hypothesis and optimal feedback control (4–6). This could also explain the lack of change in produced variability when the outward-pushing perturbation occurred after variability had increased above baseline levels (*experiment 4*): it is possible that controlled variability here returned to baseline levels (without reducing past that point) but was counteracted by the increase of error sensitivity induced by outward-pushing perturbations.

Another possibility is suggested by the fact that in reaching tasks only task-relevant variability has been observed to decrease, while task-irrelevant variability remained high (5, 53). Thus, it is possible that participants in the present experiments reduced their variability along a particular dimension, even if their overall variability increased. To explore this possibility, we calculated changes in variability along participant-specific major and minor axes of variability in the baseline phase, assuming that the minor axis of variability may be more tightly controlled (5). However, we found that participants produced similar changes in variability along the major and minor axes, suggesting that the speech motor system might control variability more globally compared with the selective regulation in certain components of movement variability observed in nonspeech motor control (54, 55). It is important to note, however, that the major and minor axes in speech production are not necessarily equivalent to the task-relevant and task-irrelevant dimensions in nonspeech control, and indeed may both contribute to task performance. Further work is needed to clarify this point.

In summary, we have shown that individuals modify their produced variability when their perceived trial-to-trial variability is altered. Decreases in perceived variability lead to increases in produced variability, likely due to loosened restrictions on variability production, particularly in individuals with inherently low variability. These changes are retained even when the perturbation is removed, suggesting that the monitoring and regulation of variability act relatively slowly. Conversely, variability also increases in response to perturbations that increase perceived variability, potentially due to

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increases in error sensitivity as participants try to correct for the perceived errors. Together, these results are consistent with recent evidence that suggests motor variability should be viewed as an important feature of how the sensorimotor system operates and learns rather than as the inevitable and unintended consequence of motor noise. Our results also highlight the importance of having a better understanding of motor variability during speech production, which has been largely overlooked in current theories and models of speech motor control.

DATA AVAILABILITY

Data and associated code are available at https://www.doi.org/10.17605/OSF.IO/STJC9. Some functions rely on additional code available at https://github.com/carrien/free-speech.

SUPPLEMENTAL MATERIAL

Supplemental Tables S1 and S2 and Supplemental Figures S1–S4: https://www.doi.org/10.17605/OSF.IO/STJC9.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

D.T., B.P., and C.A.N. conceived and designed research; D.T. performed experiments; D.T., B.P., and C.A.N. analyzed data; D.T., B.P., and C.A.N. interpreted results of experiments; D.T., B.P., and C.A.N. prepared figures; D.T., B.P., and C.A.N. drafted manuscript; D.T., B.P., and C.A.N. edited and revised manuscript; D.T., B.P., and C.A.N. approved final version of manuscript.

ENDNOTE

At the request of the authors, readers are herein alerted to the fact that additional materials related to this manuscript may be found at https://www.doi.org/10.17605/OSF.IO/STJC9 and https://github.com/carrien/free-speech. These materials are not a part of this manuscript and have not undergone peer review by the American Physiological Society (APS). APS and the journal editors take no responsibility for these materials, for the website address, or for any links to or from it.

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