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Somatosensory Contribution to Motor Learning Due to Facial Skin Deformation

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Ito T, Ostry DJ. Somatosensory contribution to motor learning due to facial skin deformation. *J Neurophysiol* 104: 1230–1238, 2010. First published June 30, 2010; doi:10.1152/jn.00199.2010. Motor learning is dependent on kinesthetic information that is obtained both from cutaneous afferents and from muscle receptors. In human arm movement, information from these two kinds of afferents is largely correlated. The facial skin offers a unique situation in which there are plentiful cutaneous afferents and essentially no muscle receptors and, accordingly, experimental manipulations involving the facial skin may be used to assess the possible role of cutaneous afferents in motor learning. We focus here on the information for motor learning provided by the deformation of the facial skin and the motion of the lips in the context of speech. We used a robotic device to slightly stretch the facial skin lateral to the side of the mouth in the period immediately preceding movement. We found that facial skin stretch increased lip protrusion in a progressive manner over the course of a series of training trials. The learning was manifest in a changed pattern of lip movement, when measured after learning in the absence of load. The newly acquired motor plan generalized partially to another speech task that involved a lip movement of different amplitude. Control tests indicated that the primary source of the observed adaptation was sensory input from cutaneous afferents. The progressive increase in lip protrusion over the course of training fits with the basic idea that change in sensory input is attributed to motor performance error. Sensory input, which in the present study precedes the target movement, is credited to the target-related motion, even though the skin stretch is released prior to movement initiation. This supports the idea that the nervous system generates motor commands on the assumption that sensory input and kinematic error are in register.

INTRODUCTION

Cutaneous afferents in the skin are a rich source of kinesthetic information (McCloskey 1978; Proske and Gandevia 2009). The role of cutaneous afferents has been investigated in human limb movement using microelectrode recording (Edin 1992, 2001, 2004) and psychophysical measurement (Collins et al. 1996, 2005; Edin and Johansson 1995). However, the prevailing view is that kinesthetic information comes largely from muscle receptors and, accordingly, attention to cutaneous afferents has been more limited (Proske and Gandevia 2009). Most of the literature on cutaneous receptors focuses on their role in pain, thermal sensation, and touch, rather than on kinesthesia (McGlone and Reilly 2010). It is unknown whether cutaneous afferents play a role in motor learning. Indeed studies of motor learning using force-field adaptation (Shadmehr and Mussa-Ivaldi 1994) in human arm movement are unable to separate the contribution of cutaneous afferents from muscle receptors because the manipulation affects all somatosensory inputs more or less equally.

Somatosensory signals arising from cutaneous afferents in the facial skin provide a means to study the role of cutaneous inputs in motor learning (Connor and Abbs 1998; Ito and Gomi 2007; Johansson et al. 1988). They are potentially valuable in understanding the kinesthetic role of cutaneous information because many orofacial structures, and notably the perioral system, lack muscle receptors (Folkins and Larson 1978; Stål et al. 1987, 1990) and cannot benefit from visual input for the control of articulatory motion. However, it is not known whether receptors in the facial skin actually contribute to motor learning and adaptation. Since the facial skin is deformed in different ways in conjunction with articulatory motions for different sounds (Connor and Abbs 1998), patterns of facial skin deformation may offer a new way of understanding sensory function in speech motor learning. In particular, since skin deformation systematically alters somatosensory input (Edin et al. 1995), changes in articulatory motion are expected in response to externally applied skin stretch.

Here we investigated the functional role of cutaneous afferents associated with facial skin deformation in motor learning. We applied facial skin deformation over a series of training trials, in an interval immediately preceding speech movements. Our approach here may be contrasted with that of other work on speech control in which researchers have examined the role of mechanical perturbations in the context of ongoing movements (Gracco and Abbs 1985; Tremblay et al. 2003). In the present study, we observe a progressive increase in lip protrusion under these conditions, which is different from previous learning studies that have observed error reduction (Shadmehr and Mussa-Ivaldi 1994). The increase was preserved as an aftereffect in trials following the end of training. Two control tests showed that the skin stretch deformation did indeed predominantly act on cutaneous afferents in the facial system. The finding documents the involvement of cutaneous afferent information in motor learning in the orofacial system. The progressive increase due to somatosensory error suggests that the nervous system produces motor commands with the expectation that sensory input correctly signals kinematic error.

METHODS

Participants and test sequence

Twenty-four native speakers of American English participated in the main experiment involving speech motor learning. Nine additional individuals participated in two separate control studies. The participants were all healthy young adults. All participants signed the informed consent forms approved either by the Yale University

Human Investigation Committee or by the Institutional Review Board of McGill University.

We examined whether patterns of lip movement in speech could be changed by deforming the facial skin lateral to the oral angle. To address this issue, we gently stretched the facial skin under computer control (see *Experimental manipulation*), to modulate somatosensory inputs in the context of a simple adaptation paradigm that entailed the repetition of a speech utterance involving lip motion. In the test, we focused on lip protrusion in the production of "w" in the training utterance, "see wood." We instructed participants to speak in their usual manner and to maintain a constant volume by monitoring a sound level meter. Each participant was asked to synchronize his/her speech utterances with audio and visual cues that helped to maintain a constant speaking rate. In total, 4 of the 24 participants showed excessive variation in the amplitude of lip protrusion movements for the production of "w" and were excluded from the analyses that follow.

In *experiment* 1, we assessed the extent to which adaptation occurred in response to the facial skin stretch perturbation. We first recorded the lip movement trajectory over the course of 20 repetitions in the absence of load. The skin stretch perturbation was then applied over the next 200 repetitions as a training phase. After the training phase, articulatory motion without the skin stretch perturbation was recorded (20 repetitions) to assess the presence of training aftereffects. We carried out a control study in which different participants repeated the same training utterance under null conditions (without the skin stretch perturbation) for the same number of trials (240). Seven individuals participated in perturbed and control conditions, respectively.

In *experiment* 2, we assessed generalization of learning by determining whether the pattern of adaptation, acquired in the context of the training task, transferred to other speech movements involving different amplitudes of lip motion. Six participants were involved in this test. We used the consonant "h" in the transfer task because it involves the weakest specification for lip configuration of any consonant in English and thus permits any generalization of prior learning to be seen with few competing requirements for the production of the "h" sound. Participants were required to produce the same training movements involving "see wood" as in *experiment* 1. Twenty repetitions of the transfer utterance " see hood" were recorded under null conditions (without the skin stretch perturbation) preceding and following the training session, to assess movement patterns prior to training and possible transfer of aftereffects as well.

We carried out two separate control tests (*experiments* 3 and 4) to evaluate the contribution of cutaneous inputs and muscle receptors to the somatosensory response to skin stretch. Muscle spindles are present in only a few muscles in the orofacial system (Folkins and Larson 1978; Stål et al. 1987, 1990). One notable exception, where muscle spindles are plentiful, is the jaw closer muscle masseter, which is immediately adjacent to the site of the skin stretch perturbation. Accordingly masseter muscle receptors may be activated during skin stretch. In a first control test (*experiment* 3), we examined whether the skin stretch perturbation induces a reflex response in masseter. We recorded electromyographic (EMG) responses in masseter under a range of skin stretch conditions and, for comparison purposes, we also recorded the stretch reflex response of masseter to a downward tap on the chin with a calibrated hammer. In a second control experiment (*experiment* 4) we recorded the position of the jaw in response to different amounts of skin stretch, to assess the possibility that the skin stretch perturbation resulted in jaw motion.

Experimental manipulation

The primary experimental manipulation involved the application of facial skin stretch under computer control, which was delivered in conjunction with the speech utterance. We programmed a small robotic device (Phantom 1.0, SensAble Technologies, Woburn, MA)

to apply the skin stretch loads (Fig. 1). The skin stretch was produced using small plastic tabs (2×3 cm) that were attached bilaterally with double-sided tape to the skin at the sides of the mouth and then connected to the robotic device through thin wires. The wires were supported by pulleys to avoid contact between the wires and the facial skin. A constant force load pulled the facial skin backward. The amplitude of the force was separately set according to the experiment. In the motor learning studies (*experiments* 1 and 2), the perturbation was timed to alter somatosensory inputs during the motion planning interval immediately preceding the target articulatory motion. We also restricted the perturbation to the interval before movement to ensure that any observed effects on lip or jaw movements were not simply the mechanical consequences of the applied load.

On each trial, two tones were presented through external speakers, separated by a 500-ms interval. The subject was asked to use the two tones as pacing signals and to start speaking 500 ms after the second tone. The load was turned on at the start of each trial (in conjunction with the presentation of visual cue and first tone) and was released at the onset of the "s" sound in "see." The duration of the force application was almost exactly 1 s, averaging 0.99 s (± 0.028 SE) over participants, and served to shift lip position backward during the period of load application. The release was timed to ensure that the load was removed before the onset of the lip movement for "w," which started about 100 ms after the beginning of the "s" sound. Thus the load had no mechanical effect on the production of lip motion for the target sound. The load amplitude was set at 1.0 N.

We focused on the skin at the sides of the mouth for a variety of reasons. The sides of the mouth are mechanically linked with the lips in terms of anatomy. Johansson et al. (1988) reported infraorbital nerve afferents with cutaneous receptive fields at this location that are activated by speech production. Stretching the facial skin at this location induced modulation of speech perception in a manner related to the associated articulatory motion (Ito et al. 2009). Skin stretch at this location also results in a compensatory reflex response that is normally evoked by unpredictable jaw position change (Ito and Gomi 2007).

In the first control study (*experiment* 3), we recorded EMG from the masseter on the left side of the face (four participants). First, we elicited a reflex response in jaw closer muscles by tapping the chin with a tendon hammer (MLA93; ADInstruments, Colorado Springs, Colorado). The onset and amplitude of the impact force were measured using a piezo-electric sensor embedded within the head of the hammer. The maximum tapping force was about 2.5 N. We used this manipulation to verify electrode placement and also to examine the latency of the jaw-jerk reflex response. We obtained EMG data from
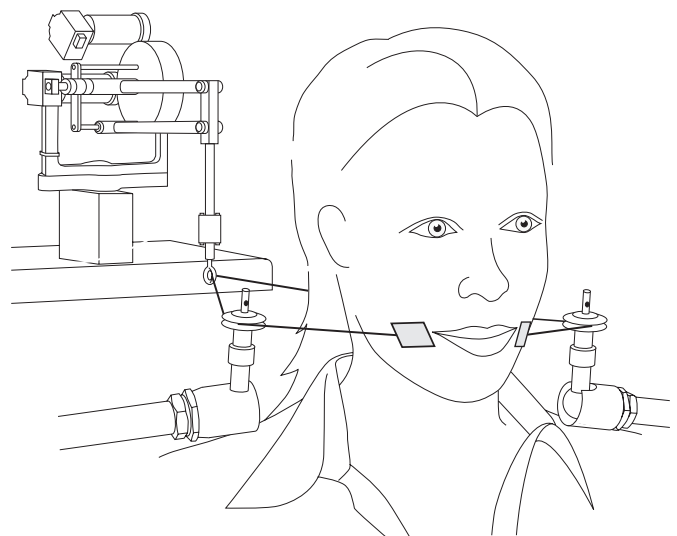

FIG. 1. Experimental set up for facial skin stretch perturbation.

20 downward taps to the chin under resting conditions. Next we examined EMG responses in the masseter muscle, using the same skin stretch perturbation lateral to the oral angle that was used in *experiments* 1 and 2 to study speech motor learning. The participants maintained steady bite force of 10% of maximum voluntary contraction with the aid of visual feedback of rectified and filtered EMG signals. Note that masseter responses to skin stretch are not observed under resting conditions. The skin stretch perturbation was delivered once background EMG activity was stable at the target level. Five amplitudes of skin stretch load (1, 2, 3, 4, and 5 N) were delivered in a random order. Twenty trials were recorded at each force level. In a second session, we used the same procedure to examine EMG responses to facial skin stretch at a location that was even closer to the masseter. In this case, the skin stretch stimulus was applied at a location immediately anterior to the EMG electrodes, that is, posterior to the original skin stretch site (lateral to the oral angle).

In a second control test with different participants (*experiment* 4), we examined whether the skin stretch perturbation altered jaw position and, in so doing, possibly activated muscle spindle afferents. Five individuals participated in this experiment. The participants were asked to hold the jaw in position for the production of "ee" as in "see." This was accomplished by having participants produce the utterance "ee" aloud and then sustain it for several seconds. Participants were instructed to stop voicing in response to a visual cue and to maintain that jaw position until a second visual cue was presented. The skin stretch perturbation was applied 500 ms after the initial cue to stop voicing. Four force conditions (0.0, 0.4, 0.8, and 1.2 N) were tested in a random order. Fifteen trials were recorded in each condition.

Signal processing

Upper lip, lower lip, jaw, and head motion and skin stretch were each sampled at 250 Hz by using the Optotrak system (Northern Digital, Waterloo, Ontario, Canada). The measurement sites depended on the experiment. Markers for the lip were put on the vermilion border at the centerline of the face. Jaw position was measured by placing a marker near the tip of a thin metal bar that was part of a dental appliance, which we attached to the mandibular teeth with a dental adhesive. Head movement measures were obtained using a small plastic plate with three markers, taped to the forehead. The markers that were used to measure facial skin stretch were put on the center of the plastic tabs at the sides of the mouth. In off-line processing after the experiment, all kinematic data were low-pass filtered at 10 Hz and head motion was removed from the articulatory position data using standard coordinate transformation procedures (Shiller et al. 1999). In the motor learning studies (*experiments* 1 and 2), we assessed the horizontal displacement of the upper lip in the production of "w" in the training task and for the production of "h" in the transfer task. The amplitude of horizontal movement was calculated as the difference between initial lip position and the maximum lip protrusion in the production of the target utterances. Initial lip position for "w" was scored at the offset of "s" in "see wood" based on our observation that lip protrusion started with the termination of the "s" sound. The offset of "s" was obtained using the acoustical analysis described in the following text. We followed the same procedure to obtain the start of the movement for "h." Maximum lip protrusion for "w" was readily identified and always coincided with the acoustically defined "w" segment. For "h," if there were no clear maxima for lip protrusion because of the relatively neutral lip configuration for the production of "h," we used the midpoint of "h" production, which was determined from the acoustic signal. In the control study involving kinematic data (*experiment* 4), we assessed position change due to the skin stretch perturbation, both in the jaw and in the facial skin lateral to the oral angle. The positions before and after the skin stretch were quantified using 100-ms time bins centered at 100 ms before the onset of the skin stretch perturbation and 100 ms immediately after. The position changes were calculated by subtracting the position before the perturbation from the position after the perturbation.

Acoustical signals were analog low-pass filtered at 8 kHz and recorded at a 16-kHz sampling rate. We focused on the first (F1) and second (F2) formant frequencies, which are the first two acoustical resonant frequencies in the speech signal and are determined by the shape of the vocal tract. F1 and F2 were extracted using Praat software at the point of maximum lip protrusion. In the production of "w" sounds, a primary vocal tract constriction between the lips and a secondary constriction near the middle of the vocal tract determine values for F1 and F2 (Stevens 1998). In general, F1 and F2 in the production of "w" are lower than corresponding formant values in "oo," in spite of a similar vocal tract configuration. Higher resonant frequencies, F3 and above, have substantially smaller amplitudes for "w" (Stevens 1998) and were not assessed quantitatively. Zero crossing rates in the acoustical signal were used to detect the start and end of "s" in "see wood" for purposes of both on-line control of the skin stretch perturbation and off-line kinematic analysis. The production of "h" was identified by the absence of acoustical power in the time interval corresponding to articulatory motion.

Electromyography (EMG) was used to measure muscle activity in the masseter. Bipolar surface electrodes (Ag–AgCl) were placed in the direction of the muscle fibers of the masseter muscle on the left side of the face. The electrode placement was determined using palpation and verified by examining EMG activity during voluntary clinching and jaw movement. We also verified electrode placement by tapping the chin to elicit a jaw reflex response. The EMG signals were amplified and band-pass filtered (30–2 kHz) with a biomedical amplifier (Grass Technologies, West Warwick, RI) and recorded at a sampling rate of 4 kHz. Rectified and filtered data were aligned at the stimulus onset and averaged in each force condition. Reflex amplitude was calculated by temporally averaging the rectified EMG signal using a 10-ms time window. This period was chosen because a 10-ms bin width had proved to be adequate in capturing perioral reflexes (Di Francesco et al. 1986; Okdeh et al. 1999) and the jaw-jerk reflex (Lund et al. 1983; Miles et al. 2004). We focused on three time periods in the reflex response, which we will refer to as JJ, R1, and R2 (see Fig. 4*A*). JJ was the interval associated with jaw-jerk reflex. To deal with individual variation in the latency of this response we used 10-ms intervals starting 5, 6, or 7 ms after stimulus onset to evaluate the magnitude of the jaw-jerk response. The R1 and R2 intervals were set at 27–37 and 52–62 ms after stimulus onset, respectively. These values corresponded to the location of peak EMG responses based on the entire data set (see Fig. 4*A*). The background EMG level (BK) was obtained in a 10-ms interval starting 20 ms before stimulus onset.

Statistical analysis

In the speech motor learning study we carried out quantitative tests using as dependent measures the amplitude of the horizontal upper lip motion in each target utterance and F1 and F2 frequencies in the acoustics. For both the kinematic and the acoustical data, we averaged on a per participant basis over blocks of 10 repetitions. We omitted the first data point (first 10 repetitions) for each participant to ensure that participants were familiar with the experimental situation. For kinematic analyses we also transformed the data to *z*-scores to reduce interparticipant variability due to amplitude differences. A one-way repeated-measures ANOVA was used to assess differences in the kinematic and acoustic data over the course of training. Regression analysis was also carried out to test for a progressive change in the training phase. To examine motor adaptation effects, we focused on kinematic differences before and after the adaptation phase and also at beginning and end of training. The former measure evaluates adaptation by examining changes in null condition movements before and after learning. The latter examines the training effect in the presence of the perturbation over the course of adaptation. We focused on whether training resulted in a nonzero aftereffect. We evaluated the

ratio of the after-training lip protrusion to the before-training protrusion, averaged over participants, as a measure of the aftereffect in the training task and also for purposes of assessing transfer of learning. We also assessed the immediate effects on movement of the onset and offset of the skin stretch perturbation. We also calculated a correlation coefficient between kinematic and acoustic measures.

In the first control test, the dependent measure was the amplitude of the EMG response in the masseter muscle. The amplitude was obtained for each participant separately by averaging rectified and filtered EMG activity during predetermined time periods (BK, JJ, R1, and R2; see Fig. 4A). We transformed the amplitude measures to z-scores on a per participant basis and averaged across participants. A two-way repeated-measures ANOVA was used to assess differences associated with the time at which the response was measured (BK, JJ, R1, and R2) and the magnitude of the skin stretch perturbation (1, 2, 3, 4, and 5 N). We carried out Bonferroni-corrected comparisons for each variable, if applicable. To assess reflex responses due to the skin stretch perturbation, we specifically focused on the difference from the background EMG level (BK) in each of the EMG measures. In the second control test, the dependent measures were the change in vertical and horizontal position of the jaw. A one-way repeated-measures ANOVA was conducted for each jaw position measure separately to assess potential differences in position across the four levels of the perturbation load (0, 0.4, 0.8, and 1.2 N). Our primary interest was the difference in jaw position between the null condition and the other three force conditions. We also calculated a correlation coefficient between the position change of the jaw and the measured magnitude of facial skin stretch.

RESULTS

The aim of these experiments was to assess the role of somatosensory information in speech learning. Somatosensory inputs were modified by stretching the facial skin under computer control in conjunction with the repetition of a speech utterance. We found that the amplitude of upper lip protrusion systematically increased over the course of training. Movement toward the new target was preserved in aftereffect trials when the load was removed after the completion of training. Figure 2A shows, for a single participant, representative examples of upper lip motion as a result of training with the skin stretch perturbation. The trajectories of lip protrusion are shown in the sagittal plane during the production of "w." The trajectories are aligned at the onset of lip protrusion. The black line shows the trajectory before training. The amplitude of horizontal motion increased slightly at the beginning of the training (green dashed line; also see Fig. 2B). At the end of the training, the horizontal amplitude was substantially magnified (blue dashed line). This change was largely maintained in aftereffect trials (red solid line). Overall, greater upper lip protrusion was observed following training.

Figure 2B shows the mean amplitude of the upper lip protrusion movement in standard score units over the course of the experiment. The symbols in the gray areas represent lip protrusion before and after the training phase in the absence of load. The line segments in the middle of the panel show the pattern of upper lip displacement over the course of training. Error bars and shaded areas surrounding the solid lines give the SE across participants. The data shown in red represent the behavior of participants trained with the skin stretch perturbation; the data in black represent control subjects. For visualization purposes, we aligned the data from control and perturbation conditions at zero before training.

We observed two effects due to the skin stretch perturbation. First, and most important, there was a progressive increase in the amplitude of upper lip displacement over the
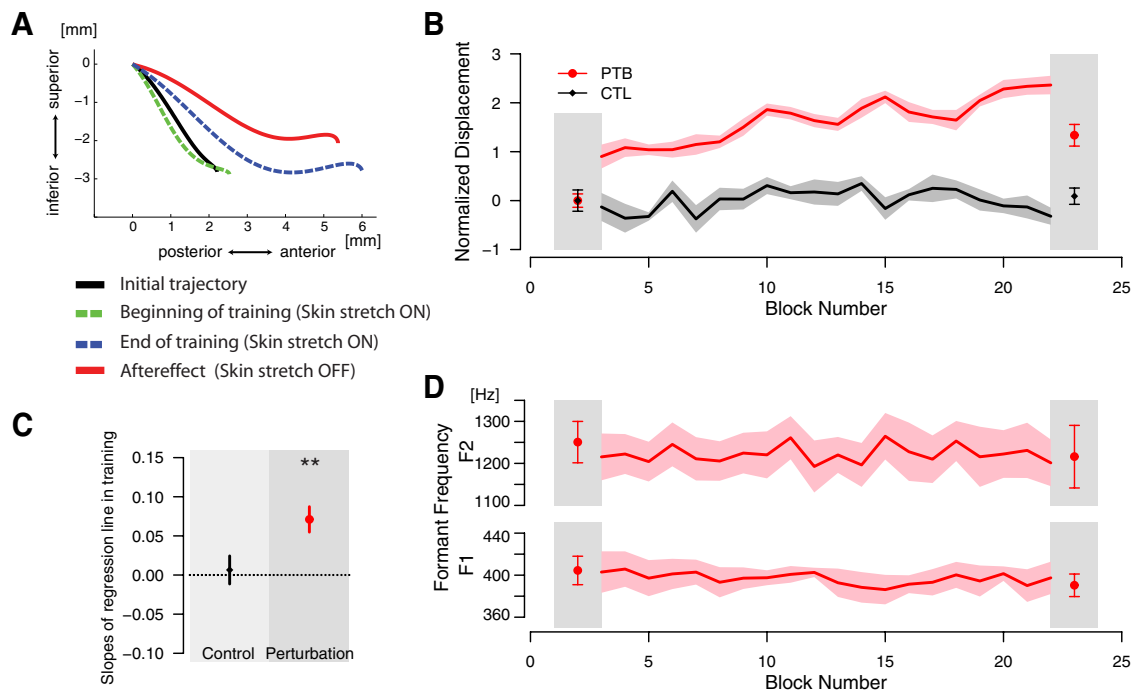

FIG. 2. A: representative sagittal plane trajectory of the upper lip for the production of "w" in the utterance "see wood." Vertical and horizontal are orientations relative to the upper skull. B: amplitude of the upper lip protrusion for the production of "w," over the course of training. Displacements were normalized by z-transformation. The dots in the shaded area showed the behavior in the null condition. Lines within the shaded area give the pattern in the training phase with SEs shown across participants. C: the slope values obtained by regression analysis for the data in the learning phase. D: pattern of first (F1) and second (F2) formants across training blocks, in the presence of the skin stretch perturbation. The dots in the shaded area show the behavior under null conditions before and after training. Lines in the shaded area show the pattern during the training phase with the SE across participants. PTB: perturbation; CTL: control.

course of the training phase (red line in Fig. 2*B*; note that absolute upper lip position also increased over training). The pattern of lip protrusion in the no-load control condition did not change over the course of training (black line in Fig. 2*B*). The progressive change in lip protrusion in the training phase was verified in a regression analysis (Fig. 2*C*). The regression was conducted on a per subject basis, using as a dependent measure the mean lip protrusion in 20 blocks of 10 repetitions each. The resulting slope, averaged over participants, was reliably different from zero in the training with skin stretch ($t_6 = 4.38$, $P < 0.005$). In the control condition, the slope was not different from zero ($t_6 = 0.36$ $P > 0.70$).

We also examined learning by assessing differences in lip protrusion between the start and end of training and also under null conditions before and after learning. Figure 3*A* shows the mean amplitude of both of these differences in standard score units. The *left panel* (training) shows an increase in upper lip displacement between the start and end of the training phase (99% confidence interval [CI], 0.067–2.85). The *right panel* (aftereffect) also shows an increase, under null conditions, between lip displacement before and after learning (99% CI, 0.24–2.42). Repeated-measures ANOVA indicated a reliable increase in protrusion [$F(1,6) = 18.55$, $P < 0.005$] that, as shown in the *right* and *left panels* of Fig. 3*A*, was similar in magnitude with the load on and off [$F(1,6) = 0.47$, $P > 0.5$]. Thus the training effect that occurs during skin stretch is preserved after removing the perturbation. The average change in lip protrusion due to adaptation (1.36 mm) was 83% of the average horizontal lip displacement for the production of "w" before training (1.64 mm). The results suggest that the motor plan was modified in a progressive manner on the basis of the skin stretch perturbation.
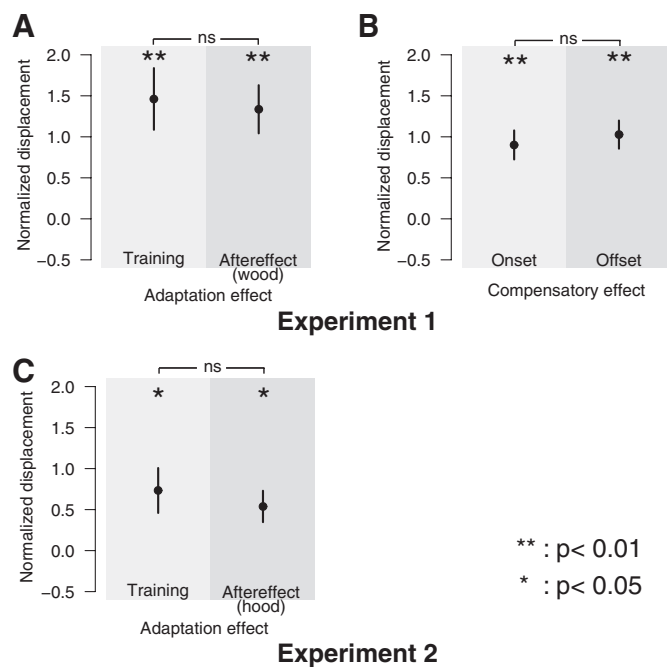
A second main effect due to the skin stretch perturbation was an abrupt compensatory change in lip protrusion. This change can be seen at the very start and end of the skin stretch sequence shown in Fig. 2*B*; the amplitude of lip protrusion increases in the trials immediately after application of the perturbation force (onset change) and suddenly decreases in the trials immediately after removal of the perturbation (offset change). The compensatory change is summarized in standard score units in Fig. 3*B*. ANOVA revealed a reliable change in lip position with the introduction or removal of load [$F(1,6) = 42.15$, $P < 0.001$]. The magnitude of the sudden lip position change was similar in the two conditions [$F(1,6) = 0.47$, $P > 0.5$]. In both cases, the overall change was reliably different from zero (99% CI, 0.24 to 1.56 for the onset change and 0.39 to 1.66 for the offset). Thus the change in lip movement that occurs in response to load thus comprises a compensatory component (shown in Fig. 3*B*) that is constant in magnitude over the course of training and an adaptive effect (Fig. 3*A*) that progressively increases over the same interval.

The finding that somatosensory inputs alter lip position in speech learning is underscored by an acoustical analysis of the perturbation group. We did not find any systematic change in either the first (F1) or the second (F2) formant frequency, measured at the peak displacement of the upper lip (Fig. 2*D*). Repeated-measures ANOVA, conducted separately for F1 and F2 frequencies, found no reliable differences over the course of 20 experimental blocks and two null blocks that preceded and followed training [$F(21,126) = 0.94$, $P > 0.5$ and $F(21,126) = 0.79$, $P > 0.7$ for F1 and F2, respectively]. Neither F1 nor F2 was correlated with the amplitude of upper lip protrusion ($r = -0.10$ and -0.03 for F1 and F2, respectively). When acoustical patterns were assessed on a per subject basis, there were individual instances in which there were acoustical differences between conditions when the skin stretch perturbation was initially applied, when it is removed, and even following the removal of all loads at the end of the experiment. However, there were no consistent patterns over subjects, nor were acoustics correlated with lip motion on a per subject basis. The absence of any systematic acoustical change may be due in part to small overall changes in vocal tract length over the course of training (2–3% increase in length) and to the possibility of compensation by the other articulatory organs to maintain acoustical outputs. The present finding is consistent with the idea that articulatory movements in speech motor learning may be controlled independent of acoustic signals (Nasir and Ostry 2008; Tremblay et al. 2003).

In a second experiment, we examined generalization of learning to other speech movements that differed in amplitude. Figure 3*C* shows differences in lip protrusion for the training utterance ("see wood") between the start and end of training and also for the transfer utterance ("see hood") under null conditions before and after learning. As in *experiment* 1, there was a change in lip protrusion between the start and end of skin stretch training (95% CI, 0.03–1.44) and likewise a change in lip protrusion before and after learning for the transfer utterance (95% CI, 0.05–1.03). However, the transfer effect is smaller than that observed with the training utterance, although the difference is not reliable statistically [$F(1,5) = 0.23$, $P > 0.6$]. The change in amplitude for "h" in the transfer utterance under null conditions was 73% as large as

FIG. 3. *A*: amplitude of training and aftereffect in repetitions of "see wood." *B*: amplitude of the compensatory effect in *experiment 1*. *C*: amplitude of the training effect following repetitions of "w" and aftereffect amplitude in the transfer task with "h." Displacements are normalized by *z*-transformation. The error bars showed the SE across participants.

the change observed during skin stretch when participants trained directly with repetitions of "w" (Fig. 3*C*).

We carried out two control tests that focus on the somatosensory inputs that are involved in the sensory modulation due to skin stretch. There are only a few muscle spindles in facial muscles, particularly in the muscles that generate lip motion (Folkins and Larson 1978; Ståhl et al. 1987, 1990). Thus the primary source of sensory information due to the skin stretch perturbation is presumably from cutaneous afferents. One exception is the jaw closer muscle masseter that has many muscle spindles. Since the masseter is close to our stimulus site (lateral to the oral angle), the skin stretch perturbation may directly stimulate masseter muscle spindles and, accordingly, the learning that we have observed could reflect this proprioceptive input. A related possibility is that the skin stretch perturbation may alter the position of the jaw and, in so doing, may activate the muscle spindles in the jaw closers. We carried out two separate control tests to assess the possibility that we were activating jaw closer muscle spindles with our perturbation.

In a first control experiment, we assessed whether the skin stretch perturbation that we used in the learning test is able to induce a jaw-jerk reflex, which is a stretch response arising from spindle afferents in the masseter (~8 ms; range: 6–10 ms; see summary in Murray and Klineberg 1984). We elicited a short-latency reflex response by tapping the chin with a calibrated jaw hammer. However, when we applied the skin stretch perturbation that we used in the test of speech motor learning and indeed even when we applied larger skin stretch forces (5 N) we obtained no evidence of this short-latency masseter response. Instead, we observed small-amplitude graded inhibitory and excitatory effects in the masseter at considerably longer latencies (~20 and 50 ms, respectively). Figure 4*A* shows the temporal pattern of masseter EMG in data from a single representative participant. The *top panel* shows the reflex response due to chin tapping. The reflex was elicited consistently in all four participants. The mean latency across participants averaged 6.2 ms (±0.78 SE). The *bottom panel* shows the EMG response due to the skin stretch perturbation. The lines in five different colors represent the averaged responses in each force condition (1, 2, 3, 4, and 5 N) for the same participant as in Fig. 4*A*. The vertical dotted line represents the timing of stimulus onset. Stretching the skin lateral to the oral angle induced reflex responses of the masseter in multiple phases. An inhibitory response occurred first between 20 and 50 ms and then an excitatory response followed between 50 and 70 ms after stimulus onset. After these responses, activity returned to the baseline level that preceded the skin stretch perturbation. This inhibitory–excitatory response is similar to the jaw reflex arising from cutaneous afferents around the lip (Di Francesco et al. 1986; Okdeh et al. 1999).

The EMG responses due to the skin stretch perturbation were examined quantitatively in an analysis in which the response was partitioned into 10-ms bins. The EMG amplitude in each bin was calculated for each participant and each force level separately. Figure 4*B* shows the averaged data in standard score units. BK, JJ, R1, and R2 refer to the periods shown in the shaded areas in the *bottom panel* of Fig. 4*A*. The data for the five force levels are represented in five different colors. Error bars show the SE across the participants. The data show inhibitory responses in R1 and excitatory responses in R2. Both responses were graded accord-
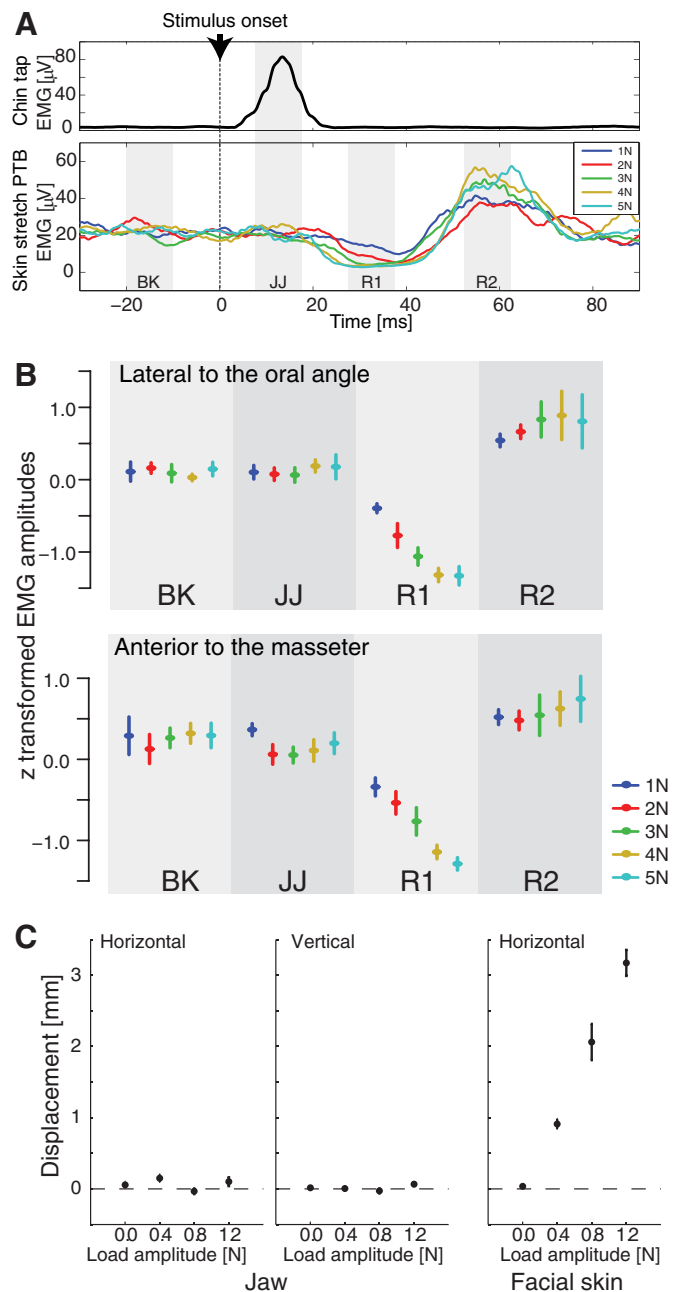

FIG. 4. *A*: representative electromyographic (EMG) response in the masseter for a single participant, due to chin tapping (*top*) and the skin stretch perturbation (*bottom*). The shaded areas show the periods for the calculation of background activity (BK), jaw-jerk reflex (JJ), and the first and second reflex responses (R1 and R2). *B*: amplitude of the EMG response due to the skin stretch perturbation. *C*: amplitude of jaw position change (horizontal and vertical) and facial skin displacement (horizontal) for 4 applied forces (0, 0.4, 0.8, and 1.2 N).

ing to amplitude of the skin stretch load. The EMG amplitude in the interval associated with the jaw-jerk response (JJ) was similar to that in BK. A two-way repeated-measures ANOVA (4 time bins × 5 force levels) showed the EMG amplitude differed among time bins [$F(3,9) = 31.49$, $P < 0.001$] and the magnitude of the effect in different time bins varied with force level [$F(12,36) = 3.03$, $P < 0.005$]. Bonferroni-corrected comparisons found a significant difference in the R1 interval in the responses to the 3, 4, and 5

N force conditions ($P < 0.05$ in all three) relative to the background level for the corresponding force conditions. In contrast, there were no reliable differences between JJ and BK ($P \geq 0.25$ for each of the five force levels). Tests for the R2 interval (relative to background EMG levels) also failed to produce any reliable post hoc effects ($P \geq 0.15$ for each force level).

A similar pattern was observed when the skin stretch perturbation was delivered immediately anterior to the EMG electrodes. Figure 4B (*bottom*) shows the mean EMG amplitudes ($\pm$SE) for this site. A two-way repeated-measures ANOVA (4 time bins $\times$ 5 force levels) found that EMG amplitude differed among the time bins [$F(3,9) = 23.13$, $P < 0.001$] and, as before, the magnitude of the effect varied with force level [$F(12,36) = 4.39$, $P < 0.001$]. The pattern of Bonferroni-corrected comparisons was likewise similar. The EMG response in the R1 interval to the 2, 3, 4, and 5 N conditions was reliably different from background activity in the same conditions ($P < 0.03$ for all four) and there was no reliable difference in activity in the JJ and BK intervals ($P \geq 0.5$ for each force level). Similarly there were no differences between R2 EMG levels and corresponding background activity ($P > 0.9$ in all cases). In summary, there is little evidence that the skin stretch perturbation elicits a reflex response in muscle spindle afferents. Instead, the observed pattern of masseter inhibition and excitation seen in the R1 and R2 intervals is similar to that which occurs in conjunction with cutaneous stimulation. This suggests that the current skin stretch perturbation stimulates cutaneous afferents. These results are consistent with the findings of Smith et al. (1988) who reported that a jaw-jerk reflex could be readily induced using percutaneous indentation to the masseter as small as 1 mm, whereas there was no excitatory reflex when the percutaneous stimulus was delivered in a motion parallel to the surface, on the skin directly above the masseter.

In a second control experiment, we examined whether the skin stretch perturbation produces a change in the position of the jaw that might elicit a proprioceptive response. We examined the change in jaw position following a skin stretch perturbation of 0.4, 0.8, and 1.2 N with that observed in a null condition (no skin stretch load). We found there was no reliable change in position of the jaw, even with a load of 1.2 N, which exceeded the one in the motor learning study. Figure 4C shows the position change of the jaw and facial skin before and after the skin stretch perturbation. The two *left panels* show the horizontal and vertical jaw position change. The *right panel* shows the amplitude of stretch in the facial skin lateral to the oral angle. The error bars show the SE across participants. The amplitude of the facial skin stretch increased with the amplitude of the applied load. In contrast, there was no reliable change in either the horizontal or the vertical position of the jaw [$F(3,12) = 1.21$, $P > 0.30$ and $F(3,12) = 1.78$, $P > 0.20$, respectively]. There was no correlation between the amplitude of facial skin stretch and the jaw position change ($r = 0.009$ in the horizontal and $r = -0.040$ in the vertical direction). Thus the skin stretch perturbation does not appear to affect jaw position in the sagittal plane, suggesting that spindle afferent input from jaw closer muscles is not altered by the skin stretch load. Based on the results of the two control tests, we conclude that the somatosensory modulation due to the skin stretch perturbation arises primarily from cutaneous afferents in the facial skin.

DISCUSSION

The principal finding of this study is that somatosensory inputs arising from facial skin deformation modify lip movement in a progressive manner over the course of training. The finding is consistent with the idea that somatosensory afferent input is involved in motor learning in speech. We also found that the newly acquired motor plan transferred at least partially to another utterance involving lip movement in the same direction. Two control studies suggested that the somatosensory information arising from the skin stretch perturbation came primarily from cutaneous afferents in the facial skin. Consequently, facial cutaneous afferents appear to contribute functionally to motor learning.

Cutaneous afferents provide information about the direction of skin stretch, as suggested by the finding that cutaneous responses of mechanoreceptors in the human arm and facial nerves show patterns of activity that vary systematically with the direction of stretch (Edin et al. 1995). A systematic dependence on the direction of skin stretch is also observed in speech sound perception, when the facial skin lateral to the oral angle is stretched (Ito et al. 2009). The present finding that lip protrusion is progressively modified as a consequence of skin stretch is consistent with the idea that skin stretch provides kinesthetic information.

We observed that over the course of training participants progressively increased lip protrusion in response to skin stretch. This is in contrast to previous studies of motor learning in arm reaching movements in which learning is reflected in movements that, over the course of training, progressively approach the original trajectory. In the present study, the somatosensory input that occurred just before movement affected sensory function during motion. The facial skin stretch was applied in a direction opposite to the upcoming movement; the resulting sensory input may have led the nervous system to underestimate lip position. Consequently, the actual motion may have been consistently evaluated as smaller than the intended one and motor commands may have been updated to progressively yield a larger movement. This idea is consistent with limb movement studies using tendon vibration in which vibration just before the actual motion induced an underestimate of displacement (Cordo et al. 1995). It may also be supported by the findings of Wolpert et al. (1995) who observed that a resistive force in arm reaching motion produced an underestimation of endpoint location compared with the movements in a no force condition.

The skin stretch manipulation offers the unusual perspective on sensory and motor elements of the adaptation process. Ordinarily, the two occur in combination, with sensory input signaling the magnitude of kinematic error and the subsequent adaptation involving changes to motor commands that result in error reduction. In the present study, sensory input due to skin stretch results in a progressive increase in lip protrusion. Our result fits with the basic idea that sensory error is attributed to motor performance error, which in turn subjects presumably attribute to the difference between actual and intended trajectories. What is novel about the present situation is that sensory input, which precedes the target movement, is attributed to the subsequent movement, even though the perturbation is actually

released prior to movement initiation. The gradual change of the lip movement trajectory over the course of training is reminiscent of the findings of Mazzoni and Krakauer (2006) who reported that, in visuomotor adaptation, implicit strategies for the use of feedback information are used even under circumstances that lead to an increase in kinematic error. In the present situation, the results support the idea that the nervous system generates motor commands on the assumption that sensory input and kinematic error are in register.

In addition to a progressive change in lip protrusion over the course of training we also observed a compensatory response that was manifest as an abrupt overshoot in the production of the target utterance. If the sensory system provided ideal inputs to the movement control system, the overshoot would have been treated as motor performance error and corrected over the course of the training. However, there were no corrections observed and indeed the magnitude of the compensatory response seemed to be maintained over training. This suggests the sensorimotor system did not take into account the sudden change in the somatosensory configuration during movement. Neglect of the sudden somatosensory change may possibly be associated with the principal adaptation effect, that is, with the progressive increase of lip protrusion over the course of training. However, its abrupt appearance (and disappearance) with the introduction (and removal) of load suggests it is different in origin. In either case, the phenomena associated with somatosensory function during movement, including sensory suppression (Chapman et al. 1987), require further investigation.

The perturbation force was released before the onset of the lip protrusion movement associated with the "w" sound. This means that the upper lip movement always occurred without external force, even during the skin stretch training phase. When questioned, some participants reported that they were aware that the force had been released before, or just after, they started to speak, but some were not aware at all of the release or its timing. Thus regardless of participants' awareness of the presence of the load, their upper lip tended to protrude more than that in control trials. This is consistent with the idea that the feedback system in motor learning is primarily a nonconscious process. Empirical support for this view comes from the observation that participants adapted to an unconscious visuomotor rotation even when it was in conflict with an explicit visuomotor learning task (Mazzoni and Karakauer 2006).

Patterns of movement generalization are central to understanding the neural organization of motor learning (Atkeson 1989). In studies of limb motor control, the general rule is that motor learning is primarily local or instance based (Atkeson 1989; Ghahramani et al. 1996; Matter and Ostry 2007). Motor learning in speech production appears to be similarly local in that speech learning fails to transfer even to utterances that involve very similar movements (Tremblay et al. 2008). The current study showed that adaptation associated with facial skin stretch transferred, although not completely to another movement in the same direction. The finding is consistent with previous work showing that generalization over changes in movement amplitude occurs only over the portion of the movement in transfer task that is similar to that previously experienced during training (Matter and Ostry 2010).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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