

# The motor cortex is involved in reflexive compensatory adjustment of speech articulation

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Although speech articulation relies heavily on the sensorimotor processing, little is known about its brain control mechanisms. Here, we investigate, using transcranial magnetic stimulation, whether the motor cortex contributes to the generation of quick sensorimotor responses involved in speech motor coordination. By applying a jaw-lowering perturbation, we induced a reflexive compensatory upper-lip response, which assists in maintaining the intact labial aperture in the production of bilabial fricative

consonants. This reflex response was significantly facilitated by subthreshold transcranial magnetic stimulation over the motor cortex, whereas a simple perioral reflex that is mediated only within the brainstem was not. This suggests that the motor cortex is involved in generating this functional reflexive articulatory compensation. *NeuroReport* 16:1791–1794 © 2005 Lippincott Williams & Wilkins.

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## Introduction

Reflexes contribute importantly to movement generation and to postural control and they are regulated precisely according to task requirements [1–3]. It has been suggested that a transcortical loop could be engaged in the sensorimotor processing that occurred in these reflexes. In humans, a study using transcranial magnetic stimulation (TMS) [4] showed that cortical pathways are involved in the long-latency stretch reflex (LLSR) for the flexor digitorum profundus, which supports the idea that the cortex is important not only in voluntary feedforward motor control [5,6], but also in sensorimotor processing involving reflex mechanisms.

In multi-articulator coordination during speech production, reflex responses are involved in the achievements of desired movements [7–10]. During production of the bilabial fricative consonant / $\Phi$ /, we have found that the upper lip rapidly, and functionally, shifts downward in response to a sudden jaw-lowering perturbation to maintain the intact labial aperture [11]. Note that this phoneme, / $\Phi$ /, is close to the sound of the English /f/, but its production does not use the upper teeth, but uses the upper lip. As a result, the production of this phoneme requires a precise control of aperture between the upper and lower lips. In the study by Gomi *et al.* [11], the upper lip muscle activity started to increase  $48.25 \pm 1.2$  ms after the jaw perturbation. This latency is longer than that in a perioral reflex (12–18 ms [8], 14–17 ms [12]) that is mediated within the brainstem alone, and shorter than the voluntary reaction time after perceiving a stimulus (jaw:  $150 \pm 13$  ms [13], finger: 154 ms [14]). Considering these facts, it can be postulated that, as in the LLSR in the limb, cortical processing is involved in this reflexive compensatory adjustment to speech articulation.

The neural pathway for generating this response, however, cannot simply be categorized as a 'cortical reflex' on the basis of response latency alone. Pearce *et al.* [15] have demonstrated that the motor cortex is not involved in the LLSR of the human masseter muscle caused by the jaw perturbation. The latency of this jaw LLSR ( $34.0 \pm 1.4$  ms) is roughly comparable to that of the reflexive compensatory response for the upper lip. This suggests the possibility that the reflexive compensatory response of the lip is mediated within a subcortical loop rather than within a transcortical loop. To determine whether the primary motor cortex is involved in reflexive compensatory adjustments to speech articulation, the current study assessed the effect of TMS over the motor cortex in the functional sensorimotor response of the lip.

## Method

Four neurologically normal individuals participated in the experiments. All signed the informed consent form of the ethics committee of the NTT Communication Science Laboratories. Two TMS experiments were performed for three of four study participants (A, B, and C). In both experiments, participants were seated on a dental chair and were asked to sustain the bilabial fricative consonant / $\Phi$ / for 2–3 s in the sentence 'kono /a $\Phi$ a/'. During the sustained consonant production, either jaw perturbation (experiment 1) or a mechanical lip stimulus (experiment 2) was supplied as explained below. A bipolar surface electrode (Ag–AgCl) was placed on the upper lip muscle (orbicularis oris superior) at the right side. The electromyographic (EMG) signal was amplified and filtered (band-pass: 20–5 kHz) with a biomedical amplifier (MME-3116, Nihon Kohden,

Shinjuku, Tokyo, Japan), and sampled at 24 kHz (DF2021, PAVEC, Hino, Tokyo, Japan). The participants monitored their muscle activity level so as to maintain it within a particular range centered at a level slightly below that for normal production of the task consonant. The lower and upper bounds were heuristically determined considering the trial variance.

A single-pulse TMS (1.5 T MAGSTIM 200, The Magstim Co. Ltd, Carmarthenshire, Wales, UK) was applied to the left motor cortex associated with the upper lip. A figure-of-eight coil was positioned tangentially in relation to the scalp. Tight strapping of the head to a headrest helped to eliminate any movement between the coil and the scalp. The stimulus site was identified so as to optimally elicit a motor-evoked potential (MEP) for the orbicularis oris superior. The identified site was in an area 2 cm anterior and 2 cm inferior to the site at which an MEP can be elicited in the first dorsal interosseous muscle and was also close to the point at which repetitive TMS induces speech disruption [16]. To check that the identified site was consistently stimulated in both experiments, we examined the MEP that was elicited using suprathreshold TMS before and after each experiment. The stimulus intensity was set 5% below the threshold level for muscle activity during production of the task utterance (40–60% for maximum output). On the basis of the fact that the latency of the MEP by TMS is around 10 ms in the orofacial muscles [17,18], the TMS onset was set at 10 ms before the start of the focused periods (indicated by the vertical dashed lines in Figs 1a and 2a) of the reflex responses. The coil position and intensity of TMS were carefully maintained throughout the two experiments.

The first experiment was designed to examine the facilitatory effect of TMS on the reflexive compensatory response to the jaw perturbation. The participant's jaw was held in the jaw perturbation system by clamping it between a chin plate and a custom-built split that was attached to the teeth [11]. Note that this apparatus results in little disruption of normal speech. The experimenter started a trial when the muscle activity level of the orbicularis oris superior entered the predetermined range in the task utterance. A step-wise perturbation (3.0 N) acted in the jaw-opening direction and was applied in 20% of the trials randomly to avoid anticipation. The total number of trials was 100, including 10 trials in each of the following conditions: PT (perturbation with TMS), PN (perturbation alone), and NT (TMS alone), and the rest for control.

To quantify the amplitude of muscle response, the rectified EMG signal during a 10-ms window (10–20 ms after TMS onset) was temporally averaged and pooled in each condition (PT, PN, and NT). This time window was defined on the basis of the fact that the MEP is induced 10–20 ms after TMS onset in the orofacial muscles [17,18]. The background muscle activity level (BK) was quantified by temporally averaging the rectified EMG signals for 10–20 ms prior to the perturbation (or stimulus) onset.

In addition, to confirm that the compensatory response was involuntary, we characterized the reaction time to the unpredictable jaw perturbation in an independent experiment. The participants ( $n=3$ : two of whom took part in the TMS experiment) were asked to protrude the lip immediately after perceiving the jaw perturbation during production of the task consonant.

The second experiment was designed to examine the facilitatory effect of TMS on the brainstem reflex in lip

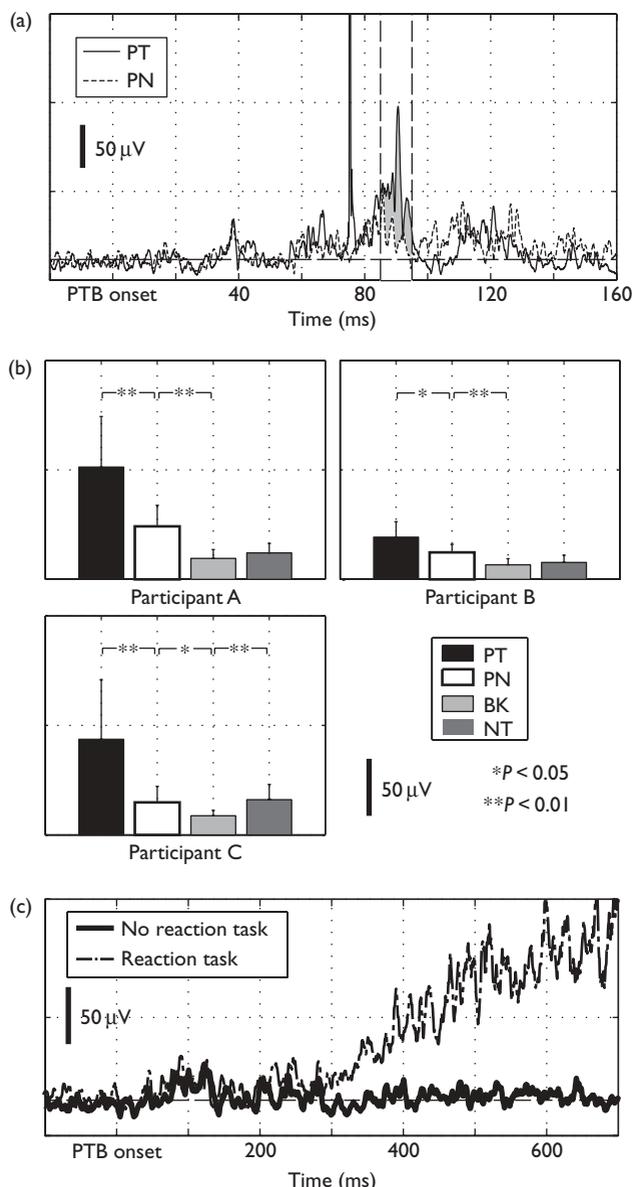
muscles. Here, the perioral reflex, which is considered to be mediated only within brainstem neural circuits, was evoked by applying a mechanical cutaneous stimulus on the right side of the vermilion using an electromagnetic shaker (Model 4810, Brüel & Kjær, Nærum, Denmark) [19]. The participants were asked to gently hold a cylinder between the upper and lower lips, while they shaped their lips for the task consonant. The cylinder was connected to the shaker. The trial onset and stimulus onset were the same as those in the first experiment. The initial acceleration peak of the stimulus was set at the intermediate value ( $100 \text{ m/s}^2$ ) in the range (60–160  $\text{m/s}^2$ ) used in [19] to avoid saturation of the reflex response activity. The participants were also asked to maintain muscle activity in the same range as in the first experiment. In this experiment, 10 trials in each condition (PT, PN, and NT) were ordered randomly for a total of 30 trials. This experiment was performed just after the first one.

## Results

For all three participants, the jaw perturbation induced a quick downward shift of the upper lip (see [11]) accompanied by a muscle EMG response (see dotted line in Fig. 1a) that served to maintain the labial aperture required for the task consonant. Thus, in addition to the mechanical linkage between the jaw and upper lip [11], a heterogenic neural linkage [8] contributes to the compensatory lip movement. The question here is what neural mechanism generates the EMG response. The three possible explanations of this response mechanism are voluntary reaction, cortical reflex, and brainstem reflex.

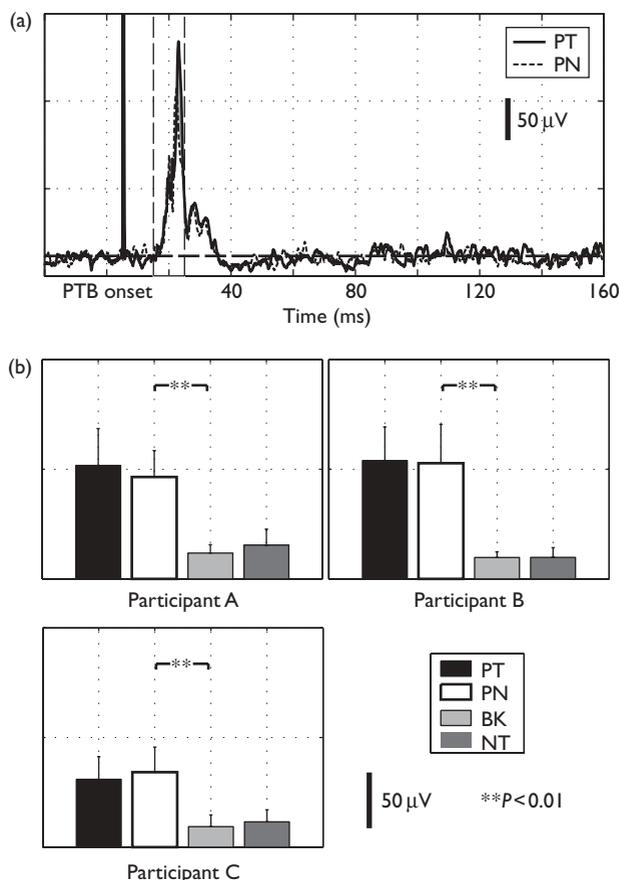
To rule out voluntary reaction, we compared EMG response latencies that resulted from the jaw perturbation with those involved in the voluntary reaction. A typical EMG response of the orbicularis oris superior in the reaction task is shown by the dashed line in Fig. 1c. The voluntary response started around 300 ms after the perturbation. The mean and standard deviation of the reaction time was  $315.7 \pm 98.4$  ms for the three participants. The shortest reaction time (137.6 ms) was comparable to that reported for jaw ( $150 \pm 13$  ms [13]) and finger muscles (154 ms [14]), and was obviously longer than the latency of the reflexive compensatory response ( $48.25 \pm 1.2$  ms [11]). Therefore, the short-latency (<100 ms) compensatory response can be regarded as nonvoluntary.

We used TMS to examine the involvement of the motor cortex in generating the reflexive compensatory response. A previous limb study [4] has shown that the subthreshold TMS facilitates the LLSR in the flexor digitorum profundus, mediated by the motor cortex. We expected that if the lip region of the motor cortex is involved in the reflexive compensatory response of the upper lip, then subthreshold TMS over motor cortex would enhance the EMG activity of the response. In a first experiment, we thus examined whether subthreshold TMS facilitates the reflexive compensatory response. The typical EMG pattern that is observed when TMS is applied during jaw perturbation (PT) is depicted by the solid line in Fig. 1a. The first sharp peak 75 ms after the perturbation onset was an artifact induced by current spread due to TMS. Compared with the response without TMS (PN), an increase in EMG activity started 10 ms after TMS onset and continued for roughly 10 ms (the shaded area). The 10 ms latency is comparable to the MEP latency associated with TMS for the orofacial muscles



**Fig. 1** (a) Typical reflexive compensatory response with or without TMS in rectified and averaged EMGs of the orbicularis oris superior (participant A). The solid line denotes the response in the 'perturbation with TMS' (PT) condition; the dotted line gives the response in the 'perturbation alone' (PN) condition. The horizontal dashed line denotes the background EMG activity level (BK). Time zero is the jaw perturbation onset, and the TMS pulse was applied 75 ms after that. The two vertical dashed lines indicate 10 and 20 ms after TMS onset, respectively. The shaded area (gray) indicates the EMG activity enhanced by TMS. (b) Means and standard deviations ( $n=10$ ) of the response amplitude in three cases [PT, PN and TMS alone (NT)] with BK. (c) Rectified and averaged EMG signal of orbicularis oris superior in the reaction task (dashed line) superimposed on the response in the no-reaction task (solid line) (participant A). TMS, transcranial magnetic stimulation; EMG, electromyogram; PTB, perturbation.

(the upper lip:  $11.8 \pm 1.8$  ms [18], jaw:  $10.5 \pm 1.5$  ms [17]). The quantified response amplitudes in all cases (PT, PN, BK, and NT) are summarized in Fig. 1b. TMS consistently enhanced the reflexive compensatory response in all participants, as shown by the difference between PT and PN cases ( $t$ -test: participants A and C,  $P < 0.01$ , participant B,  $P < 0.05$ ), whereas there was no significant enhancement of muscle



**Fig. 2** (a) Perioral reflex by mechanically stimulating the vermillion of the upper lip with TMS (PT: solid line) and without TMS (PN: dotted line) for participant A. The horizontal thin dashed line denotes the background EMG activity level (BK). The two vertical dashed lines denote times 10 and 20 ms after TMS onset, respectively. The shaded area (gray) indicates the EMG activity enhanced by TMS. (b) Means and standard deviations ( $n=10$ ) of response amplitude in the three cases [PT, PN and TMS alone (NT)] with the background EMG level (BK). TMS, transcranial magnetic stimulation; EMG, electromyogram; PTB, perturbation.

activity in NT compared with BK ( $t$ -test:  $P > 0.20$ ) for participants A and B (upper two graphs of Fig. 1b). In the NT case for participant C, the muscle activity was slightly enhanced (bottom graph of Fig. 1b). The enhanced EMG activity in PT, however, was considerably greater than that in NT [difference between (PT minus PN) and (NT minus BK)], suggesting that the facilitatory effect was the primary determinant of the enhanced EMG activity in PT. In summary, these facilitations suggested that the transcortical pathway significantly contributes to the production of the reflexive compensatory response.

One could claim that this facilitatory effect is due to brainstem activity change rather than the motor cortex. To determine which neural excitability (brainstem or motor cortex) is dominant, we examined, in a second experiment, the TMS excitability of the perioral reflex that is known to be mediated only within the brainstem [8,12,19]. Note that the subthreshold TMS over the motor cortex does not enhance muscle activity produced by reflexes that are mediated within the spinal cord alone [4]. The perioral reflex was elicited  $17.75 \pm 0.92$  ms after stimulus onset (PN: the dotted line in Fig. 2a, which is almost overlapped by the solid line),

which is relatively comparable to the results of previous studies (12–18 ms [8], 14–17 ms [12]). Even when the same TMS as in the first experiment was applied, the perioral reflex (PT: thick line) was not enhanced as shown in Fig. 2a. The quantified response amplitudes in Fig. 2b also indicate that there was no significant difference between PT and PN under these conditions ( $t$ -test:  $P > 0.40$  for all participants). This indicates that the facilitatory effect of this subthreshold TMS fully depends on the neural excitability in the motor cortex, and not that of the brainstem. Taken together, these results suggest that the primary motor cortex is involved in generating the reflexive compensatory response observed during speech articulation.

## Discussion

Although numerous studies of the limb control mechanisms have demonstrated that cortical reflexes, such as the LLSR in the flexor digitorum profundus [4], contribute to 'servo action' in stabilizing a posture and/or in a reaching movement [1,2], little is known about whether this kind of reflex is utilized in the orofacial system. Furthermore, if it exists, its role in human speech articulation is unclear. Human anatomical data [20] indicates that the facial motor nucleus, that contains the lip motor neurons, receives direct projections from the motor cortex. This direct connection was also physiologically supported by the small latency variation in MEP onset in the lip muscle induced by TMS [18]. No study, however, has examined how this direct corticobulbar tract functions in coordinated orofacial movements.

In this study, we used TMS to examine whether the motor cortex is involved in generating the lip compensatory response observed during production of the bilabial fricative consonant. Our results indicate that the facilitatory effect of TMS on the response reflects the enhancement of neural excitability not in the brainstem but in the motor cortex. This strongly suggests that the primary motor cortex is involved in generating the reflexive compensatory response. Namely, this transcortical compensatory reflex as well as a volitional control mechanism is employed in the motor control of speech articulation.

For the bilabial stop consonants (/p/ and /b/), similar reflexive compensatory upper lip muscle responses were observed using unpredictable perturbations to the lower lip [8] or jaw [7,9]. Although the involvement of the supra-bulbar pathway has been suggested [8] because of the medium latency (22–75 ms), the neural mechanisms have not been clarified yet. From a functional viewpoint, reflex responses act to maintain the intact lip posture for closing or making a constriction. It can be therefore inferred that reflexive articulation for bilabial stop consonants, as well as that for bilabial fricative consonants, is mediated within the cortex. As noted in the introduction, because latency alone is not sufficient evidence for the identification of a cortical reflex, further investigation is required to clarify the involvement of the transcortical loop for various reflexive muscle responses during speech articulation.

## Conclusion

By demonstrating the facilitatory effects of TMS to the cortex on the reflexive compensatory response in lip

muscles during labial speech production, we suggest that the primary motor cortex is involved in its generation. The possibility of a TMS-induced enhancement of the reflex response at the motor neuron level was rejected because we found no facilitatory effect of the same TMS on the brainstem reflex. High-level computation in the cortex would greatly contribute to the organization of complex sensorimotor coordination among articulatory organs for the robustness of speech tasks.

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