DECOUPLING FUNCTIONAL UNITS IN SPEECH PRODUCTION USING AUDITORY STARTLE

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ABSTRACT

Functionally defined neuromuscular structures, or modules (e.g., agonist-antagonist pairings), may be decoupled when elicited by a startling auditory stimulus (SAS), revealing possible lower-level functional units. The current study examines lip kinematics in SAS-induced responses and uses a 3D FEM biomechanical model to simulate the temporal interaction between facial muscles used in speech. Results show that SAS-elicited bilabial production is subject to displacement discontinuity (i.e., velocity change); this displacement discontinuity appears to be accounted for by the face model simulation results showing temporally decoupled coordination across neuromuscular modules. These findings suggest possible lower-level neuromuscular modules for speech movements that may correspond with long-described articulators in speech production.

Keywords: speech modules, temporal decoupling, startle, Artisynth

1. INTRODUCTION

Speech production, like other motor behaviours, requires spatiotemporal coordination between independently controllable neuromuscular structures in the body [11]. Direct observation of these structures, however, has been elusive. Microstimulation findings [1, 2, 10] reveal that movements may be viewed as comprising modularized neuromuscular systems organized in spinal and peripheral areas. We may infer that such structures exist in human behavior, and that these in some cases appear to be phylogenetically encoded, as in swallowing or locomotion [5]. In other cases, as in acquired speech movements, such structures are thought not to be fully innate, but to be developed through accumulated practice and experience [8]; however, even such ontogenetically acquired or adapted organizations, once established, may act as semiclosed functional units, effectively reducing dimensionality of the speech motor control space [6].

Recent work has shown that even canonical semiclosed neuromuscular systems are susceptible to disruption by a startling auditory stimulus (SAS). For example, in limb motor control, the well-known triphasic activation pattern (agonist $[AG_1]$ – antagonist $[ANT]$ – agonist $[AG_2]$) may be observed to occur with great regularity in targeted movements, such as extension and flexion [7, 2]. Forgaard et al. [6] use a SAS paradigm to examine whether this triphasic pattern may be disrupted to reveal deeper structures; their findings show that when a SAS is presented at the onset of AG_1, the response time for ANT and AG_2 is accelerated. These targeted movements with AG-ANT forces may thus in fact result from coordination of lower-level structures [4].

Using the startle paradigm to investigate speech movement sequencing, Stevenson et al [12] show that a prepared CV syllable can be released at a short latency by a SAS, and that the kinematics of SAS-induced responses are more compressed than those of control responses. In the present study, we reexamine the lip kinematics from [12] with regard to temporal coupling between the lower lip and jaw; the same sequences are implemented in a computer 3D model (www.artisynth.org) [9] to simulate lip mechanics and kinematics, in order to uncover the biomechanics of the underlying neuromuscular structures. The present study is designed to address the question of whether kinematic anomalies induced by SAS may be explained in terms of a disruption of temporal coupling between structures within a neuromuscular modular framework.

2. RE-EXAMINATION OF SAS-INDUCED LIP KINEMATICS

Kinematic data from Stevenson et al. [12] Experiment 2 were re-examined. While a range of lower lip kinematic variation was observed, two schematic types were identified (Figure 1). Type I shows a single continuous upward movement of the lower lip during compression (Figure 1 top). The corresponding velocity illustrates a smooth and positive acceleration during lip compression. No disconti-

nuity was observed during deceleration and during the opening of the lower lip. Type II responses, on the other hand, demonstrate two distinct events from closure to opening, which in turn translate into displacement discontinuity (i.e., as manifested by a velocity change) during opening (Figure 1 bottom). Among the 171 trials analyzed in Stevenson et al. [12], the proportion of Type I startle responses was notably dominant (97.5 %) in control responses, while in SAS-induced responses a four-fold increased in Type II responses was observed (from 2.5 % to 10.3 %).

Figure 1: Two representative trials from Stevenson et al. [12] Experiment 2 showing schematic lower lip displacement (black) and corresponding velocity (red). Type I (top): undisrupted lip compression and opening; Type II (bottom): disrupted opening
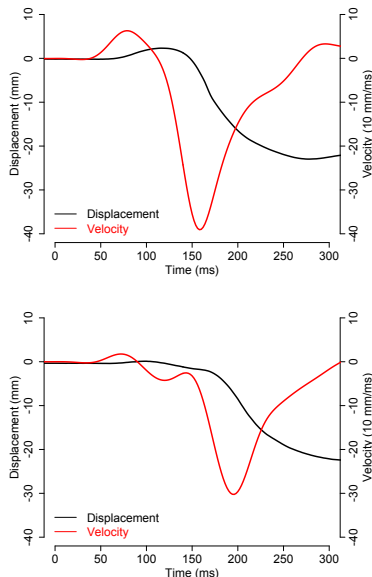
The present study uses the Artisynth simulation platform to provide biomechanical simulations of the syllable [ba] to test whether Type II responses in SAS-induced responses may be the result of inter-effector temporal decoupling. Simulations were implemented using a face and vocal tract model built in Artisynth, a 3D simulation platform using finite element methods (FEM) to simulate head and neck biomechanics [9].

3. SIMULATION SET-UP

Muscles are activated up to a maximum stress level (kPa), as indicated in Table 1. To trace the lower lip movement trajectory, the coordinate location of a node located in the center of the lower vermilion

(yellow dots in Figure 2) was logged.

Table 1: Maximum muscle stress (kPa) used for a closure posture and voluntary [ba]

	rest position (plain closure)	voluntary opening for [ba]
OOM	30	43
OOP	30	43
MENT	20	30
RIS	20	30
BUC	20	30
ABD, MH, GH	–	80

Figure 2: The Artisynth face model in plain closure (left) and open (right) positions
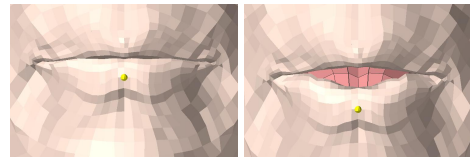
Muscles used to simulate the production of [ba] include orbicularis oris peripheral (OOP), orbicularis oris marginal (OOM), mentalis (MENT), risorius (RIS), buccinator (BUC), and the jaw-lowering muscles, including anterior belly of the digastric (ABD), mylohyoid (MH), and geniohyoid (GH) . At the beginning of the simulation, the mouth has been set in a closed position. Muscle activations are set to generate a closure of [b] and an opening movement for the vowel [a]. In each simulation attempt, no muscles are activated during the first 200 ms; this stipulation is made to ensure that no gravity effects are present. From 200 ms to 400 ms, muscles are activated to make a complete closure. Perioral muscle activations for voluntary [b] (OOM, OOP, MENT, RIS, and BUC) start at 600 ms and reached their force peak at 680 ms. Concurrently with this force peak of the perioral muscles, the jaw-lowering muscles start to activate, reaching their maximum force at 840 ms.

Table 2: Maximum muscle stress (kPa) used for startle reflex and amplified voluntary [ba]

	startle reflex	amplified opening for [ba]
OOM	50	60
OOP	50	60
MENT	36	44
RIS	36	44
BUC	36	44
ABD, MH, GH	80	80

As was shown in Stevenson et al. [12], a SAS induces a reflexive activity, followed by an accelerated and amplified EMG activity for the voluntary [ba]. The averaged results from Stevenson et al. [12] Ex-

periment 2 were used to calculate the EMG activity peaks for the startle reflex and the amplified voluntary onset. Amplified EMG activity in startle trials were calculated in proportion of EMG activity in control trials. The values were transformed into percentages, which were in turn translated into a measure of maximum muscle stress (kPa), where 100% corresponds to 100 kPa. The settings used to simulate SAS-induced responses are presented in Table 2. The startle reflex on the perioral muscles is followed by accelerated EMG activity with the jaw-lowering muscles either time-locked at 30 ms prior to the peak or at the peak of the voluntary EMG activity.

4. RESULTS

Type I lower lip movement consists of a continuous upward movement from the lower lip. It is presumed that this compression between the lips is the result of the EMG activity elicited by the SAS. Specifically, when a SAS is presented, a startle reflex for the perioral muscles is induced, followed by augmented and accelerated EMG activity for the voluntary movement. In our simulation attempts, these two EMG events overlap by 50 ms. When the jaw-lowering muscles are set to be active 30 ms before other perioral muscles reach their activation peaks for the voluntary movement, the superposed muscle activity from these two EMG events results in a continuous lower lip movement during compression (i.e., no velocity change, as in Figure 3 top). Lip compression is followed by an opening movement with no velocity change from negative to positive. The current simulation results show that advanced jaw movements yield smooth lip kinematics, even when following a SAS-induced reflex.

Type II lower lip trajectories are seen when the jaw-lowering muscles are activated concurrently with the onset of the voluntary [ba], as was performed in normal voluntary [ba] production. In Type II movements, the compression peak is reached earlier; this result follows from the fact that, due to their early activation, the jaw-lowering muscles worked against the muscle force creating compression (Figure 3 bottom). While working against the accelerated lip EMG, the increase of the jaw-lowering muscles starts to lower the lips. As the lip-closing muscles attain their stress peak, they arrive at an equilibrium with the jaw-lowering muscles. Consequently, the lowering of the jaw is compromised, since the two competing muscle forces are pulling the lips in two different directions (i.e., the lips are compressing against each other while the jaw-lowering muscles are pulling the lips apart). After the equilibrium,

Figure 3: Lower lip displacement (black) and corresponding velocity (red) for Type I (top) and Type II (bottom) movements. See text for detailed muscle settings.
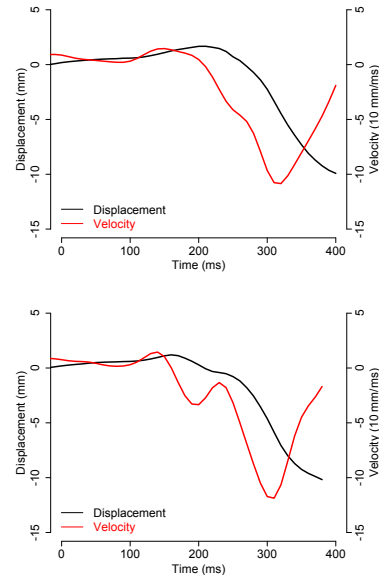
the increasing activation force from the jaw-opening muscles continues to lower the jaw and lips, leading to a velocity change during the lowering, as observed in Figure 3 (bottom). In this simulation, the jaw-lowering muscles are accelerated and advanced early to align with the onset of the voluntary [ba]. With this early activation of the jaw-lowering muscles, the lips are compressed and the opening movement is interrupted due to two conflicting muscle forces. This result is best accounted for by appealing to the decoupled relationship between the lip-closing and jaw-lowering muscles in the context of a SAS. When a SAS is presented, the activation of the lip and the jaw muscles may be subject to interruption. The temporal coordination between lip compression and jaw lowering is decoupled. As a result, a disrupted opening movement is induced.

5. DISCUSSION

As revealed in SAS-induced arm movements, coordination across agonistic and antagonistic muscles associated with the same movement task may be disrupted [6]. The SAS-induced disruption between AG_1 and ANT suggests that the programming and the execution of different muscles within a relatively closed structure associated with a single motor task may be disrupted. Similarly, the above results suggest that lip-jaw coordination in bilabial stop production is also subject to disruption by a SAS. In

SAS-induced responses, there was a higher prominence of Type II responses, in which lower lip displacement undergoes a change in velocity during opening. The increased number of Type II responses in SAS-elicited speech movements suggests that a SAS may break down the temporal coordination between different lower level neuromuscular structures associated with the same speech task.

Artisynth simulation results further support the proposal that the interruption and change in velocity during lip opening arise due to the decoupled relationship between the neuromuscular modules associated with bilabial closure and opening. With amplified muscle activity, when the jaw-lowering muscles are time-locked with the peak force of the perioral muscles (as in voluntary [ba] production), disrupted movements are observed during the opening of the bilabial burst (Type II movements). On the other hand, when the initiation of the opening occurs concurrently with the onset of the second activation for the voluntary movement, early activity from the jaw-lowering muscles is canceled out by the increasing force from the perioral muscles (Type I movements). In particular, the increased number of Type II movements supports the view that the lower-level neuromuscular subgroupings governing jaw lowering and lip closing associated with bilabial stop production are not in the same timing relationship as in Type I movements.

The observed kinematic disruption and simulation results reported here suggest, first, that the neuromuscular structures associated with speech movements, such as the oral closure for [ba], are likely built up from lower-level neuromuscular primitives; second, that these primitives correspond to interpretable, naturalistic functions (e.g., jaw lowering, lip closing) [3]; and third, SAS may be used to disrupt these larger structures and reveal lower-level structures.

6. ACKNOWLEDGEMENT

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