



Effects of mastication on human somatosensory processing: A study using somatosensory-evoked potentials



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ABSTRACT

The aim of the present study was to investigate the effects of mastication on somatosensory processing using somatosensory-evoked potentials (SEPs). Fourteen healthy subjects received a median nerve stimulation at the right wrist under two conditions: Mastication and Control. SEPs were recorded in five sessions for approximately seven minutes: Pre, Post 1, 2, 3, and 4. Subjects were asked to chew gum for five minutes after one session in Mastication. Control included the same five sessions. The amplitudes and latencies of P14, N20, P25, N35, P45, and N60 components at C3', frontal N30 component at Fz, and P100 and N140 components at Pz were analyzed. The amplitude of P45–N60 was significantly smaller at Post 1, 2, 3, and 4 than at Pre in Control, but not in Mastication. The latency of P25 was significantly longer at Post 2, 3, and 4 than at Pre in Control, but not in Mastication. The latency of P100 was significantly longer at Post 2 than at Pre in Control, but not in Mastication. These results suggest the significant effects of mastication on the neural activity of human somatosensory processing.

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1. Introduction

Previous studies have demonstrated that mastication affects human cognitive processing (Onyper et al., 2011; Hirano and Onozuka, 2015). The reaction time (RT) is an important measure of cognitive processing in investigation of sensorimotor performance (Schmidt, 2000), and is defined as the time from the stimulus onset to the response, which includes components such as stimulus evaluation and response selection (Doucet and Stelmack, 1999). Mastication has been shown to accelerate RT (Chu, 1994; Hirano et al., 2008, 2013; Sakamoto et al., 2009a,b, 2015). However, based on the behavioral data of RT only, it remains unclear whether mastication affects stimulus input processing and/or response output processing in the central nervous system.

Somatosensory-evoked potentials (SEPs), which are obtained by time-locked averaging electroencephalography (EEG) with high temporal resolution, have been used to evaluate somatosensory processing (i.e. ascending central processing). SEPs are elicited by stimulating peripheral nerves, such as the median nerve at the

wrist or posterior tibial nerve at the ankle. P14, N20, P25, N35, P45, N60, and frontal N30 are then determined as short-latency components after median nerve stimulation, and P100 and N140 components are identified as long-latency components (Nakata et al., 2003, 2011; Kida et al., 2004). Previous studies have evaluated SEPs as an index of neural activity in somatosensory processing. For example, the amplitudes of short-latency SEPs were found to be attenuated during voluntary movement (Nakata et al., 2003; Kida et al., 2006a,b), passive movement (Nakata et al., 2003), movement preparation (Kida et al., 2004), muscle relaxation (Wasaka et al., 2012), and interfering tactile stimuli (Jones, 1981; Kakigi and Jones, 1985), while the amplitudes of long-latency SEPs were increased during voluntary movement (Nakata et al., 2003) and movement preparation (Kida et al., 2004). These findings suggest that neural activity for somatosensory processing is inhibited by movement-related activity in the early stage of processing, but is enhanced in the late stage.

In the present study, we utilized SEPs, and aimed to investigate the effects of mastication on human somatosensory processing. We hypothesized that mastication influences arousal, and alters the amplitude and/or latency of SEPs from the resting control condition. The level of arousal was adjusted according to neural activity in the brainstem (Moruzzi and Magoun, 1949), and the neural pathways basic to the cortical arousal response are known as the ascending reticular activating system (ARAS). We consider the

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ARAS to be affected by mastication because rhythmic mastication is generated by a central pattern generator (CPG) in the brainstem (Nakamura and Katakura, 1995; Yamada et al., 2005; Lund and Kolta, 2006). Our previous studies used event-related potentials (ERPs), and showed the significant effects of mastication on human cognitive processing (Sakamoto et al., 2009a,b, 2015). To the best of our knowledge, however, the effects of mastication on somatosensory processing have not yet been examined by recording SEPs. We assumed that if mastication influences arousal, the amplitude and/or latency of SEPs will be affected by mastication. That is, the amplitudes of some SEP components on mastication may be increased or maintained with repeated sessions, whereas those under the resting control condition may decrease. Several previous studies reported the effect of habituation on SEPs, and showed the reduction of the amplitudes of SEPs, which are generated from the primary somatosensory cortex (SI) (Tomberg et al., 1989; Inoue et al., 2002; Ozkul and Uckardes, 2002; Restuccia et al., 2011) and secondary somatosensory cortex (SII) (Inoue et al., 2002), with repeated sessions. In addition, we inferred that the latencies of some SEP components on mastication would be shortened or maintained with repeated sessions, while those under the control condition would be delayed. This is because our previous studies demonstrated that mastication accelerated the peak latency of some components in ERPs (Sakamoto et al., 2009a, 2015).

2. Materials and methods

2.1. Subjects

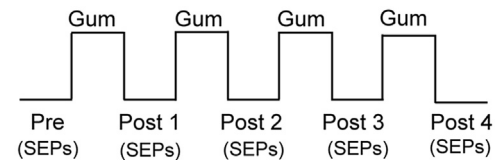
Nineteen normal right-handed subjects (three males and sixteen females; mean age 22.6 years, range 19–36) participated in the present study. None of the subjects had a history of neurological or psychiatric disorders. Informed consent was obtained from all subjects; however, the aim of the experiment performed was not explained in order to avoid any effect of bias. The study was approved by the Ethical Committee of the National Institute for Physiological Sciences, Okazaki, Japan, and Nara Women's University, Nara City, Japan.

2.2. Experiment procedure

The experiment consisted of two conditions: Mastication and Control, with each being performed on a different day. Half of the subjects began with the Mastication condition and the other half with the Control condition. The Mastication condition comprised five sessions of recordings: Pre, Post 1, Post 2, Post 3, and Post 4. Each session took approximately seven minutes. Subjects were asked to chew gum for five minutes at a relaxed self-pace after one session. There were four gum-chewing intervals (Fig. 1A) in total. The gum was removed from the mouth during EEG recording periods. A special gum base that was odorless and tasteless was prepared (CAT21 Chewing Pellet, NAMITEC Co., LTD., Osaka, Japan), and was made of polyvinyl acetate, wax, and polyisobutylene based on Japan food hygiene laws. Each gum was packed. The Control condition included the same five sessions (Pre, Post 1, Post 2, Post 3, and Post 4); however, subjects were instructed to relax without chewing gum in each interval (Fig. 1B).

In order to record SEPs, the electric stimulus used was a constant current square-wave pulse delivered to the right median nerve at a rate of 0.5 Hz. The stimulus duration was 0.2 ms, and the stimulus intensity was sufficient to produce a slight, but definite twitch of the thumb. Subjects were instructed to keep their eyes open and look at a small fixation point positioned in front of them at a distance of approximately 1.0 m. Two hundred stimuli were applied in each session.

(1) Mastication



(2) Control



Fig. 1. Protocol for Mastication and Control conditions. SEPs were recorded in five sessions under each condition. In Mastication, subjects were asked to chew a gum base that was odorless and tasteless during the intervals between sessions for five minutes. In Control, subjects were instructed to relax without gum-chewing during the intervals.

2.3. EEG recordings and analysis

SEPs were recorded with Ag/AgCl disk electrodes placed on the scalp at Fz, Cz, Pz, and C3' (C3' was 2 cm posterior to C3), according to the International 10–20 System. Each electrode was referenced to linked earlobes. In order to eliminate eye movements or blinks exceeding 100 μ V, an electrooculogram was recorded bipolarly with a pair of electrodes placed 2 cm lateral to the lateral canthus of the right eye and 2 cm above the upper edge of the right orbit. Impedance was maintained at less than 5 kohm. All EEG signals were collected on a signal processor (Neuropack MEB-2200 system, Nihon-Kohden, Tokyo, Japan). The bandpass filter of the amplifier was 1–1000 Hz. The analysis time was 100 ms including a prestimulus baseline period of 10 ms for P14 and N20, and 300 ms including a prestimulus baseline period of 30 ms for P100 at Pz. The sampling rate was 5000 Hz. The peak amplitude for P14 at C3' was measured using baseline-to-peak as the far-field potential. The amplitude of N20 at C3' was also measured using baseline-to-peak, not peak-to-peak, as the near-field potential, because the amplitude of P14 can be easily affected by various factors, such as the upper limb position. As for the subsequent components (i.e., P25, N35, P45, and N60 at C3' and frontal N30 at Fz), the peak-to-peak measurement was used. This is because the sequential component would be affected by the previous component. For example, the amplitude of N35 is easily affected by the amplitude of P25, and N35 often shows a positive rather than negative potential, when the baseline-to-peak measurement is used. We followed the same analysis methods as employed in many previous studies (Hoshiyama and Sheean, 1998; Nakata et al., 2003, 2011, 2015; Rossi et al., 2005; Wasaka et al., 2012). The peak amplitude of P100 was measured using baseline-to-peak at Pz as the long-latency component, because a clear previous component was not defined at Pz. The sequential N140 component was measured using peak-to-peak. Therefore, the amplitude was defined as P14, N20, N20–P25, P25–N35, N35–P45, P45–N60, frontal N30, P100, and P100–N140. The peak latencies for the individual SEP components were determined using a measuring scale on the Neuropack system with visual inspection, and shown as P14, N20, P25, N35, P45, N60, frontal N30, P100, and N140.

The peak amplitude and latency data of each SEP component were separately subjected to an analysis of variance (ANOVA) with repeated measures using Condition (Mastication vs. Control) and Session (Pre, Post 1, Post 2, Post 3, and Post 4) as within-subject factors. The data of five subjects were excluded because they did not match the criteria for submission to ANOVA with repeated

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