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Peak morphology and scalp topography of the pharyngeal sensory evoked potential

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Abstract

The initiation of the pharyngeal stage of swallowing is dependent upon sensory input to the brainstem and cortex. The event-related evoked potential provides a measure of neuronal electrical activity as it relates to a specific stimulus. Air-puff stimulation to the posterior pharyngeal wall produces a sensory evoked potential (PSEP) waveform. The goal of this study was to characterize the scalp topography and morphology for the component peaks of the PSEP waveform. Twenty-five healthy men and women served as research participants. PSEPs were measured via 32 electrode cap (10-20 system) connected to SynAmps2 Neuroscan EEG System. Air puffs were delivered directly to the oropharynx using a thin polyethylene tube connected to a flexible laryngoscope. The PSEP waveform is characterized by 4 early and mid-latency components peaks: an early positivity (P1), and negativity (N1), followed by a mid-latency positivity (P2), and negativity (N2). The early positive peak P1 is localized bilaterally to the lateral parietal scalp, the N1 medially in the fronto-parietal region, and the P2 and N2 with diffuse scalp locations. Somatosensory and premotor regions are possible anatomical correlates of peak locations. Based on the latencies of the peaks, they are likely analogous to somatosensory and respiratory related evoked potential peaks.

Keywords

sensory evoked potential; electroencephalography; pharynx; deglutition

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This study was completed at the evoked potentials laboratory in the Department of Physiological Sciences, University of Florida

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Introduction

The pharyngeal region of the upper airway (UA) serves as a conduit for air and for bolus material during breathing and swallowing, respectively. Sensory deficits in the UA are implicated in disorders of swallowing and cough (1-4), and in obstructive sleep apnea (5-7). Sensory receptors in the UA serve as “triggers” for the airway protective mechanisms of swallowing and coughing, and may act in a feedback loop responsible for maintaining airway patency during sleep (8-10). As such, it is important to understand the sensory components of the UA, both generally and within specific anatomic regions.

Studies in decerebrate, vagotomized, paralyzed and ventilated cats demonstrate significant responses of glossopharyngeal nerve receptors to transient negative and positive pressures applied to the UA (11, 12). In the human pharynx, sensory receptors from the pharyngeal branches of the glossopharyngeal and vagus nerves innervate the oropharyngeal wall (13). These cutaneous receptors appear to be sensitive to pressure changes within the UA. Daubenspeck and colleagues (14) demonstrated UA contribution to evoked potentials elicited via negative mouth pressure in humans using a laryngeal mask airway (LMA) whereby short duration negative pressure pulses applied with and without the LMA in place produced significant amplitude and latency differences in somatocortical peak response. This study indicated UA receptors contribute significantly to the mouth elicited cortical evoked potential (CEP) waveform.

The respiratory related evoked potential (RREP) waveform, elicited by inspiratory mechanical loads, is characterized by an early negativity (Nf) at about 43 ms, an early positivity (P1) at about 63 ms, and a subsequent negativity (N1) at 109 ms (15). While the functional underpinnings of the Nf peak remain speculative and have only been reported with RREP recording (16), the P1 peak is most likely an indicator of arrival of sensory information to the somatosensory cortex (17), while the N1 peak is associated and augmented with attention to sensory stimuli (18). Based on their latency and scalp location, Chan and Davenport (2008) suggest the P1 and N1 RREP waveforms are analogous to somatosensory P50 and N100 peaks respectively, occurring at approximately the same latency and scalp locations (17).

Mechanical stimuli applied to the lips, buccal cavities, tongue, faucial arches, soft palate, nasal cavity, and nasopharynx will evoke a cortical sensory response (19-24). Gow and colleagues (24) reported positive and negative peaks with latencies ~80ms and ~150 ms, respectively, following transient electrical stimulation to the pharynx. Hummel et al. (20) described late peaks (>200ms) in response to air puff stimuli applied to the nasal and nasopharyngeal regions. Fujiu and colleagues (19) described early far-field potentials as well as intermediate near field potentials associated with mechanical stimulation of the anterior faucial pillars. Chan and Davenport (15) recorded a positive peak at 63 ms and negative peak at 85 ms when they used air puffs delivered to the buccal mucosa of the mouth. Yoshida and colleagues (23) used magetoencephalography (MEG) to record sensory responses from air-puff stimuli delivered to the medial and lateral soft palate; this yielded a positive peak at 130 ms. Teismann et al. (25) measured the MEG response to air-puffs delivered via a nasogastric tube passed into the pharyngeal region; they found bilateral activation of the caudolateral

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primary somatosensory cortex. Movement-related cortical potentials during saliva and water swallows were characterized by an early positivity following contraction of the submental muscle group associated with swallowing (26). Additionally, studies using functional magnetic resonance imaging (fMRI) demonstrated that air puff delivery to the unilateral or bilateral peritonsillar region elicits activation of primary sensory and motor cortices, cingulated gyrus, insula, and thalamus (27, 28).

These studies demonstrate: 1.) cortical activity directly resulting from UA sensory stimuli, and 2.) cortical activation associated with mechanosensory stimulation specifically of oral and pharyngeal regions. Sensory receptors located in the posterior oropharyngeal region play an important role initiating and modulating an effective swallow. Swallowing disorders, or dysphagia, can occur due to primary sensory deficits (2, 3). This region is also implicated in the pathogenesis of OSA (5, 6). The morphological characteristics of the CEP response to mechanosensory stimulation in the posterior oropharynx are not known. The goal of this project was to identify the component peaks and morphological characteristics of the CEP response to air puff stimuli applied directly to the posterior oropharyngeal wall. It was hypothesized that the application of air puffs focally to the medial posterior oropharyngeal mucosa would result in CEP waveforms characterized by peaks analogous to other sensory modalities (i.e., respiratory and somatosensory).

Material and Methods

This study was approved by the Institutional Review Board at the University of Florida.

Subjects. Twenty-five healthy adults (8 men, 17 women) participated in the study. The mean age was 20.76 +/− 3.43 years. All subjects self reported no history of cancer in the head or neck regions, neurologic disease, or chronic respiratory disease. None of the participants were current smokers. Participants were asked to refrain from caffeine for 12 hours prior to participating in the experiment. The experimental procedures were explained upon arrival to the laboratory, and all participants provided written informed consent to participate.

Pulmonary function test

All participants were screened with a pulmonary function test (PFT). Forced vital capacity (FVC) was measured at least 3 times for each participant. The participant was instructed to respire normally through a filtered mouthpiece with nose clips in place. Following 3-4 rest breaths, they were asked to provide a deep inspiration followed by a forced expiration. Instructions were based on the American Thoracic Society standard for spirometry testing. The forced expired volume within 1 second (FEV_1) and the FVC were recorded (Jaeger Toennies, Medizintechnikmit System) and the ratio of FEV_1/FVC was used for analysis. All subjects had an FEV_1/FVC ratio greater than 75% predicted. The R5 resistance was measured with impulse oscillometry (Jaeger Toennies, Medizintechnikmit System) and the mean R5 resistance was 3.00 +/− 0.67 $cmH_2O{\cdot}L^{-1} \cdot s$, within the predicted normal range for all participants.

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Subject Preparation

A 32-electrode Neuroscan Quickcap™ based on the International 10-20 system was positioned on the participant's head and connected to the SynAmps^2 Neuroscan System. Conducting gel was applied through each electrode in order to establish scalp contact and maintain impedance levels below 5kΩ. Bipolar electrodes were placed on the skin above and below the left eye for recording vertical electro-oculogram (VEOG) activity. Synamps^2 amplifiers (Neuroscan, El Paso, TX) and SCAN version 4.3 acquisition software (Neuroscan, El Paso, TX) were used to record the EEG signal onto a desktop computer. The sampling rate was set to 1000Hz per channel with a recording bandpass of DC to 200Hz. The EEG activity was referenced to linked earlobes. SCAN version 4.3 analysis software (Neuroscan, El Paso, TX) was used for data analysis.

Participants were instructed to relax and sit comfortably in a chair with the neck, back and arms supported. A mouthpiece with a polyethylene tube was placed in the mouth, and a flexible laryngoscope was inserted through the tube (Figure 1). The laryngoscopic images were displayed, but not recorded, on a computer screen. Both the laryngoscope and computer were components of the JEDMED StroboCAM II® system (JEDMED Instrument Co., St Louis, MO). In this manner, the laryngoscope allowed for visualization and verification of tube placement for air puff delivery. The laryngoscope itself was covered with a hygienic sheath (Slide-On® Sheath for Sensory Tesing, Medtronic Xomed, Inc., Jacksonville, FL) that has a small port through which the air puffs were delivered. The port was connected to an air tank via tubing (outer diameter 2.5mm, inner diameter 2.0mm) connected to a solenoid valve that delivered air puffs to the laryngoscope port. The air tank was in-series with a digital manometer (Fluke 713 30G, John Fluke MFG Co-Inc, Everett, WA), allowing for monitoring and control of air puff pressure (Figure 1). A manual trigger system that provided an electrical output was used to initiate the data sample collected by the computer simultaneously with air-puff delivery. Air puffs were delivered in sets of 50 per trial, taking approximately 2-3 minutes per trial.

Protocol

Air puffs were delivered through the laryngoscope port, inserted and anchored through the mouthpiece. In order to place the delivery port, participants were instructed to relax their jaw and tongue, and to breathe through the mouth. Participants were then asked to sustain the vowel “/i/” that raised the soft palate and allowed for insertion of the tube through the posterior oral cavity and into the oropharyngeal region. The delivery port was situated immediately in front of, but not touching, the paramedial posterior oropharyngeal wall. Placement was verified visually on the computer screen. Once in place, preliminary air puffs were delivered in order to establish that the participant could feel the air puff specifically on the posterior oropharyngeal wall. It was not acceptable to feel generalized pressure in the throat, and the air puff pressure was adjusted until participants indicated they felt a discrete air puff stimulus on the posterior oropharyngeal wall. The pressure at the air tank was recorded for each subject, and ranged from 15 – 30 cm H_2O. Air pressure dissipates as it moves through tubing, through the sensory testing sheaths, and is delivered out of the sensory testing sheath into the oropharynx. As such, the delivery pressure at the output of the sensory testing sheath ranged from 1- 4 cmH_2O. Monitoring of the air tank output

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pressure ensured that air puff delivery pressure was maintained at a consistent level throughout the study for all participants. Care was taken such that there was not ambient movement of the mouthpiece associated with air puff delivery. Four trials of 50 air puffs per trial were presented for a total of 200 stimuli. Air puffs were delivered with an inter-pulse interval of at least 3 seconds, and varied beyond that according to any extraneous movements introducing noise into the EEG signal by the participant. Subjects rested for at least 1 minute between trials at which time they were allowed to drink water if desired.

Data Analysis

Five-hundred millisecond epochs (100ms pre stimulus and 400ms post stimulus) were sampled when the air puff stimulus was triggered. During offline analysis using SCAN 4.3 software (Neuroscan, El Paso, TX) each data frame was reviewed and the inclusion criteria for epochs were 1) no VEOG eyeblink activity, and 2) no change of EEG activity exceeding 50μV. Responses to stimuli that were confounded by artifacts were excluded from analysis. A minimum of 190 air puff epochs were averaged to obtain the pharyngeal sensory evoked potential (PSEP) waveform. The peak latencies were measured from the time of the stimulus onset, and amplitudes were measured from baseline to peak for each component.

In order to identify the component peaks of the PSEP, the averaged waveforms for each subject were displayed individually on a computer screen. Concurrent with the waveforms, a 2-D head model corresponding temporally to the waveform and depicting all recording electrodes was displayed (Figure 2) using source localization software (Scan 4.3). The software was used to determine EEG amplitude at and between the electrodes. Concurrent visualization of the waveform and 2-D head model allowed for identification and scalp localization of the positive and negative component peaks. The area of greatest positive or negative EEG amplitude corresponding with the respective waveform peak was termed the ‘hot spot’ for that peak. The latency and amplitude of the electrode closest to the hot spot was then recorded for descriptive statistical analysis.

Results

Pharyngeal sensory evoked potentials related to air puff stimuli consisted of 4 early-to-mid latency component peaks (Figure 3). The first positive peak P1 occurred at a mean latency of 58.44 +/− 10.96 (SD) ms and was followed by a negative peak N1 at 90.56 +/− 19.14 (SD) ms. The second positive peak P2 occurred at 122.56 +/− 22.43 (SD) ms, and the following negative peak N2 at 165.00 +/− 29.19 (SD) ms (Figure 4). Means and standard deviations for peak amplitudes are in the Table 1.

Hot spot scalp locations for each component peak are represented in Figure 5. The P1 peak was post-centrally lateralized in all participants, with approximately equal distribution between the left (n = 10) and right (n = 11) sides. In the remaining 4 participants, the P1 hot spot was located at the Cz, Pz or CPz midline electrodes. The N1 hot spot was located pre-centrally in 20/25 participants; it was recorded from the post-central Pz (n = 3) electrode and CPz (n = 2) in the remaining participants. N1 was located at midline in 17/25, and lateral in the remaining 8 participants; 6 were lateralized to the left and 2 to the right. Scalp location of the P2 electrode was diffusely located between participants, with 9 pre-central, 5 central

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and 11 post-central electrodes. P2 was found in 7 left, 10 midline, and 8 right electrodes. Similarly diffuse, N2 was post-centrally located in 14 participants, centrally in 5, and pre-centrally in 6. Twelve of 23 N2 hot spots were located at the midline, 8 lateralized right and 5 to the left.

Discussion

Air puff stimuli delivered to the posterior oropharyngeal wall produced consistent evoked potential waveforms in all subjects characterized by two positive (P1, P2) and two negative (N1, N2) component peaks. Thus, the PSEP waveform is characterized by four component peaks that can be defined as follows: 1.) a positivity (P1) occurring between 35.52ms - 80.36ms (95% CI) post stimulus onset; 2.) a negativity (N1) occurring between 52.28ms – 128.84ms post stimulus onset; 3.) a second positivity (P2) occurring between 79.68ms – 165.44ms post stimulus onset; and 4.) a second negativity (N2) occurring between 108.62ms – 222.38ms post stimulus onset.

The earliest positive peak, P1, occurred at a mean latency of 58.44ms and is likely analogous to both the somatosensory P50 and RREP P1. In their 2008 study, Chan & Davenport used air puffs to record the somatosensory P50 and N100 from the hand and oral cavity. They found a P50 mean latency of 58.24ms for the hand, and 63.72ms for the mouth. While it is surprising the latency was not shorter than that found for the hand given the inherent difference in the length of the afferent pathway, the latency for the PSEP P1 peak reflects the time including triggering of the stimulus, stimulus transduction through the tube, as well as afferent fiber tract properties (length and conduction velocity). When the stimulus delivery through the tube duration of approximately 20ms is accounted for, the resulting latency difference between the hand P50 and PSEP P1 can be most easily attributed to differences in the length of the afferent fiber tracts; sensory tracts ascending from the pharyngeal region have a much shorter distance to the central nervous system versus those from the hand. This assumes similar peripheral afferent conduction velocity and central neural pathways, which is reasonable given the comparable nature of the mechanical stimuli (air puffs). The 5.28 ms latency difference for the mouth P50 versus the PSEP P1 may be due to the stimulus transduction time, possibly because of the lower pressure of the air puffs (25 cmH2O in the current study, versus ~20cmH2O in the former study), or because of the presence of a mouthpiece in the current study where it was not used in the former. However, the mouthpiece in the current study remained stable for the duration of each trial and would not presumably have an effect on the event-related recorded response to stimulation delivered much more posteriorly. As well, there are likely significant differences in mechanosensitivity in the two regions and this may related to the latency difference. Mu and Sanders (13) found relatively dense conglomerations of sensory nerve terminals in the lateral (more than 10 terminals/cm^2) and medial (between 5 and 10 terminals/cm^2) posterior oropharynx. While the specific density of mechanosensitive nerve terminals has not been defined for the lateral buccal mucosa, Cordeiro and colleagues (29) found the cheek to be among the least sensitive oral areas based on Semmes-Weinstein monofilament (AKA Von Frey hair) testing. Thus, it may be that decreased mechanosensitivity and subsequent increased stimulus transduction time of the buccal mucosa accounts for the slightly longer mouth P50 latency.

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The P50 and RREP P1 peaks indicate initial arrival of the sensory stimulus at the somatosensory cortex (30). According to the scalp topography procedures of hot spot identification used in the current study, the P1 peak was located predominately in post-central lateral recording sites (Figure 5). While EEG recording electrodes can measure changes in electrical activity at the scalp only, based on the 10-20 system of electrode configuration the source of the P1 peak was measured in the area most likely overlaying the lateral parietal somatosensory primary (SI) or association (SII) cortex. This scalp location would then corroborate the findings of fMRI studies that identify activation of primary and secondary somatosensory areas with air-puff stimulation to the peritonsillar region (27, 28).

Following the P1 peak, the negative N1 PSEP peak occurred at a mean latency of 90.56ms from stimulus onset, and about 32ms following the P1 peak. Based on the latency from P1, and source localization of this peak, it is most likely analogous to the somatosensory N100, and RREP N1 occurring at mean latencies of 96.06ms for the hand, 85.23ms for the mouth, and 109.42 for the RREP. Latencies from P50/RREP P1 to the N100/RREP N1 are 38ms and 22ms, and 45ms, respectively. Scalp topography procedures indicated the PSEP N1 peak reflects midline, pre-central peak; this is consistent with the general finding that the N1/N100 peak is a vertex peak that is consistently recorded at the Cz electrode. Both the somatosensory N100 and RREP N1 peak may be modified by attention to the stimulus (18), and has been referred to as a “gating” peak, with reduction in peak amplitude occurring in response a second stimulus in a paired-stimulus paradigm (e.g., two air puffs, 500 ms apart) (31). This is hypothesized to be related to filtering of redundant sensory information to preserve cognitive and attentional resources; reduction in gating (undergating) is associated with ‘sensory overload’ in some psychiatric disorders (32). It would be interesting to examine if the gating effect is present in the oropharynx similar to somatosensory and respiratory-modality experiments.

The second positive P2 peak was present in all participants and had a mean latency of 122.56ms from stimulus onset, about 64ms following P1. Source localization indicates a largely heterogeneous distribution of the P2 peak. Verkindt and colleagues (33) hypothesized the P2 peak resulted from 2 peak generators, with one being more frontal than the other; if this is the case it may explain the diffuse P2 distribution found in this study. Perhaps between subjects the 2 generators were variable in amplitude strength, leading to greater variability in identification of the hot spot electrode. Mean latency for the RREP P2 is170 ms (17). P2 has been studied extensively in the auditory modality; reports on auditory evoked potentials (AEP) indicated average P2 latency between 150 – 250ms (34) from stimulus onset. Yoshida et al. (23) used magnetoencephalographic (MEG) procedures to study mechanosensation of the soft palate. Using air puff stimuli, they found a positive peak at 130ms in the bilateral temporal region following stimulus presentation. While no earlier peaks were reported in that study, the latency and localization of the peak suggests it is associated with stimulus processing and not initial arrival at the somatosensory cortex (e.g., a P1 peak). Additionally, given the relatively low amplitude of peaks found in the current study versus those reported for other somatosensory regions and the respiratory–related studies, it may be that the peak detection threshold requirement in the Yoshida study (resting activity mean +/− 2 standard deviations) was too stringent to allow for detection of earlier component peaks. Like the N1 peak, P2 is thought to be associated with attention and

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processing of a stimulus, as well as sensory gating (34-36). It is modified with higher level cognitive processing (37), and emotional state (38).

The last component peak consistently identified is the N2 negativity that occurred at a mean latency of 165ms from stimulus onset. Source localization procedures indicated this peak is predominately post-centrally located. Peak latency for the N2 peak resulting from mechanosensory stimuli delivered to the hand is 135ms, and for the RREP is 211ms (17). Many studies have examined the N2 AEP peak that has a mean latency between 100 and 250ms. The AEP N2 is hypothesized to be related to sensory inhibition processing and decision processing for behavioral responses (39, 40). It is unclear whether comparison to the AEP N2 is appropriate given modality differences; however it is possible that in the current study N2 was related to active inhibition of some airway defense mechanism, such as swallow, cough or gag. We have previously reported the urge-to-cough resulting from air puff stimulation to the posterior oropharyngeal region (41) from a subgroup of subjects reported for the present study. While cough expression occurred in only 5/9 subjects, all nine participants reported a noticeable urge-to-cough on at least one air-puff trial; in total, 67.6% of air puff trials were associated with an urge-to-cough. Thus, if the PSEP N2 is functionally similar to the AEP N2, it may be that PSEP N2 is related to participants actively suppressing cough during the trials.

Of note, the method employed in the current study for identifying component peaks selects only one hot spot electrode for each of the peaks identified. This methodology was chosen in part because of the poor spatial resolution of scalp EEG recordings. As such, presence of event-related activity at other electrode locations is not precluded with the identification of a hot spot electrode. Rather, the hot spot only indicates a general scalp recording location closest to the strongest dipole generating the positive or negative amplitude measure. Therefore, based on these results conclusions cannot be drawn regarding laterality of sensory responses. We can only (1) identify the presence of component peaks overlaying general cortical regions, and (2) discuss latency aspects as event-related EEG recording has excellent temporal resolution.

In summary, the oropharyngeal region is densely populated with sensory receptors, and is important in the overlapping functions of upper airway patency and swallowing. This study demonstrates the ability to evoke sensory responses analogous to those reported for other sensory modalities. Given the functional significance of this region, several additional questions emerge from these results: would the responses be different for patient populations including those with obstructive sleep apnea, or neurogenic dysphagia? Does this system exhibit gating of repetitive sensory stimuli as other sensory systems do? How would anesthetic to the oropharyngeal mucosa impact the PSEP response? Future studies should examine these questions.

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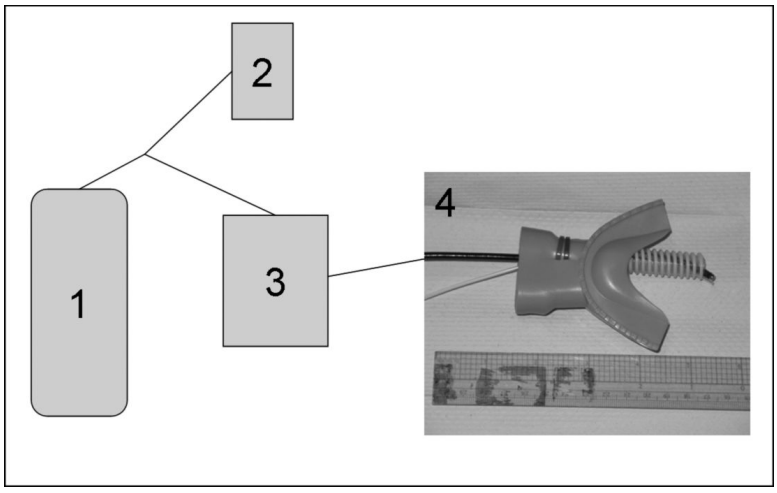

1.

Air-puff delivery equipment set-up. 1 = Pressurized air tank; 2 = Digital manometer; 3 = Trigger box with solenoid valve; 4 = Mouthpiece used for routing and placement of the flexible laryngoscope.

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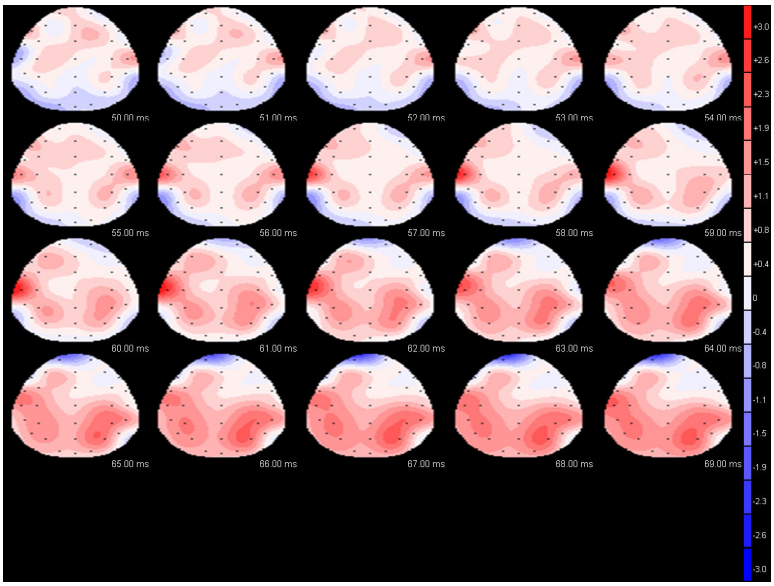

2.

A representative 2-D series for the development of the P1 peak over all recording scalp electrodes. The final picture in the series identifies the peak that corresponds to the highest peak amplitude recorded from the waveform.

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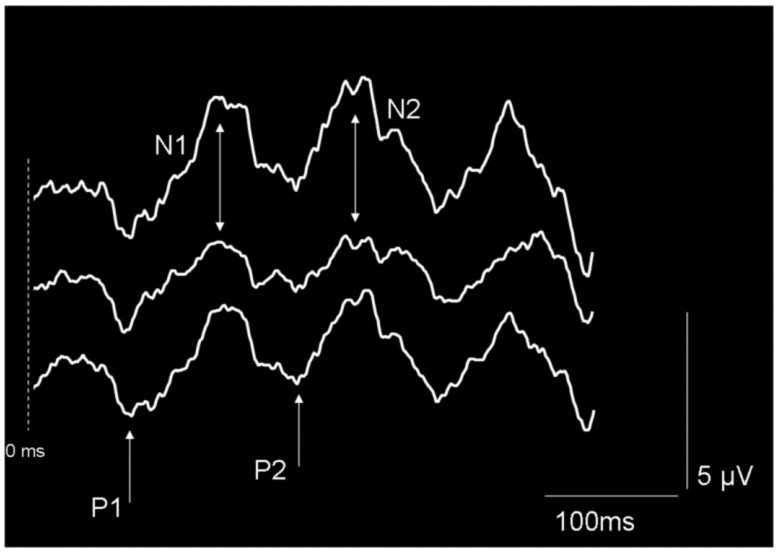

3.

A representative waveform from 3 recording electrodes of one participant. Upward deflections indicate negative peaks, and downward deflections are positive. The arrows indicate what peak (P1, N1, P2, or N2) is identified.

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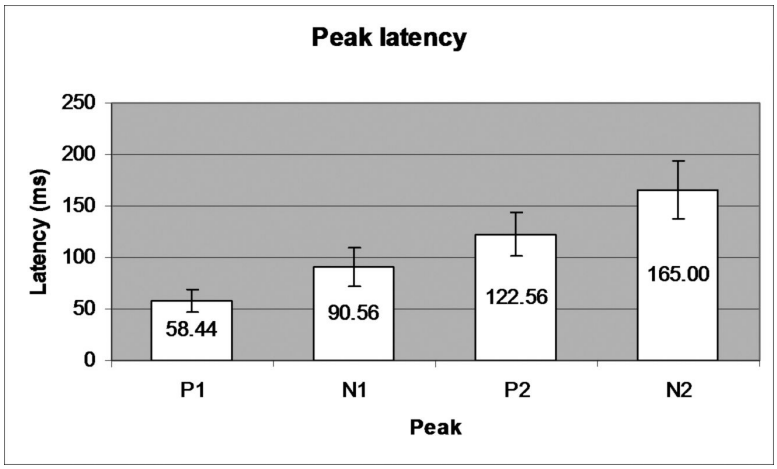
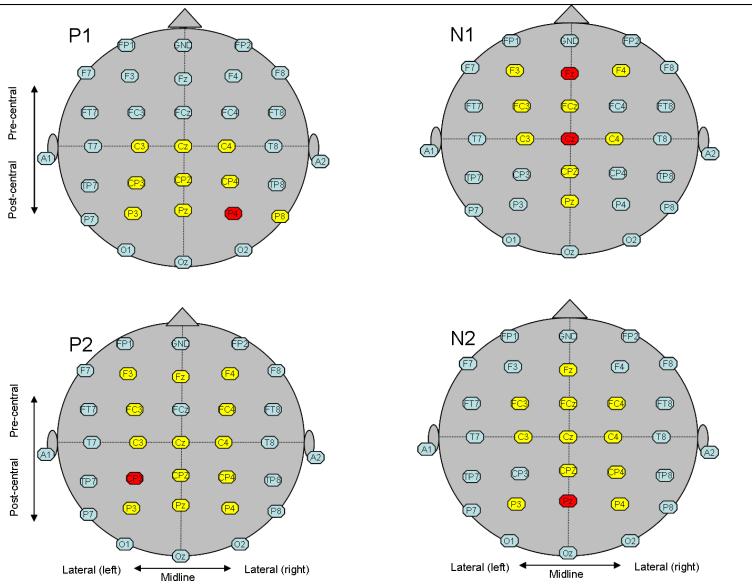


4.

Graphical depiction of the mean latency (y-axis) for each of the 4 peaks (x-axis). Means are provided, and error bars indicate +/− 1 standard deviation.



5.

Electrodes identified as ‘hot-spot’ recording electrodes for each of the 4 peaks. Yellow indicates the electrode was identified as the hot spot in at least 1 participant; red indicates the electrode(s) that were identified most often as the hot spot for each peak. Arrows on the left side of the figure indicate pre- or post-central electrodes; arrows on the bottom indicate lateral or midline electrodes.

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Table 1

Mean and standard deviation (SD) values for the PSEP component peaks

	Amplitude Mean (SD)
P1	1.16 (0.85)
N1	−1.20 (0.76)
P2	1.01 (0.47)
N2	−1.29 (0.82)