## SEP Response

The primary SEP peaks of interest were the N20 and P24-27 ([**Figure 4**](http://www.plosone.org/article/info%3Adoi/10.1371/journal.pone.0036407#pone-0036407-g004) **and** [**Table 1**](http://www.plosone.org/article/info%3Adoi/10.1371/journal.pone.0036407#pone-0036407-t001)). These peaks were recorded from an electrode site contralateral to the median nerve stimulus location (CP4). The respective average latencies of the N20 and P24-27 peaks relative to stimulus onset were 19.0 ± 0.6 ms and 24.4 ± 0.8ms (MOTOR LOW), 18.9 ± 0.6ms and 24.2 ± 0.8ms (MOTOR HIGH), 19.1 ± 0.6ms and 24.4 ± 0.8ms (SENSORY LOW), 18.8 ± 0.5ms and 24.4 ± 0.8ms (SENSORY HIGH). The average N20 to P24-27 peak-to-peak amplitudes for each of the four task conditions were 3.6 ± 0.5µV (MOTOR LOW), 4.6 ± 0.7µV (MOTOR HIGH), 3.4 ± 0.6µV (SENSORY LOW) and 4.2 ± 0.5µV (SENSORY HIGH). With respect to the latency of the N20 and P24-27 peaks, there were no significant main effects of stimulus intensity (F1,10 = 0.80, p = 0.3911 and F1,10 = 0.51, p = 0.4933, respectively) or task (F1,10 = 0.31, p = 0.5884 and F1,10 = 0.23, p = 0.6400, respectively) and no significant interaction between intensity and task (F1,10 = 2.57, p = 0.1399 and F1,10 = 0.82, p = 0.3866, respectively). There was a significant main effect of stimulus intensity on the peak-to-peak amplitude difference between the N20 and P24-27 waves (F1,10 = 12.70, p = 0.0051), no significant main effect of task (F1,10 = 0.00, p = 0.9498) and no significant interaction between task and intensity (F1,10 = 0.00, p = 0.9685).

## ERP Response

EEG responses were averaged relative to the response time (mouse click onset) and therefore this analysis was restricted to comparing the LOW and HIGH stimulus for the MOTOR task condition. Overall, there was a consistent large pre-movement negativity (PreN) followed by a positivity evoked just prior to the mouse click (PreP) that were maximal at the Cz cortical site ([**Figure 5**](http://www.plosone.org/article/info%3Adoi/10.1371/journal.pone.0036407#pone-0036407-g005)). The average PreN-PreP peak-to-peak amplitude was significantly greater following the HIGH intensity stimulus compared to the LOW intensity stimulus (HIGH: 15.8 ± 1.5 µV and LOW: 11.9 ± 1.4 µV; F1,10 = 14.01, p = 0.0038). Following the HIGH intensity stimulus, the average latencies of the PreN and PreP responses were -125 ± 7 ms and -30 ± 9 ms, respectively, relative to the response time. Following the LOW intensity stimulus, the average latencies of the PreN and PreP responses were -135 ± 14 ms and -39 ± 9 ms, respectively, relative to response time. PreN and PreP peak latencies were not significantly different between the HIGH and LOW intensity stimuli (F1,10 = 0.53, p = 0.4834 and F1,10 = 0.56, p = 0.4733, respectively.)

## Faster Reaction Time Is Not Due To Changes in the Latency of Somatosensory Processing

The finding that reaction time is shorter based on elevated stimulus intensity is not novel. Numerous studies, across various domains, using multiple modalities, have demonstrated the important role of stimulus intensity on an individual’s reaction time [[15](#_ENREF_15),[19](#_ENREF_19),29,[30](#_ENREF_30)]. However, the attempt to map the mechanism of such a response using EEG following an electrical stimulus is an important and unique step in characterizing the sensorimotor pathways involved in the generation of augmented responses. In the current study, we did not find evidence of a statistically augmented SEP latency based on stimulus intensity, despite the significant reduction of reaction time and response latency. However, there was a statistically significant different in the SEP amplitude based on stimulus intensity, which reinforces that the high stimulus intensity would have recruited a greater number of afferent axons, resulting in a greater volume of activity at the level of the somatosensory cortex. The complementary lack of change in SEP latency is likely a consequence of the relatively short distance and number of intervening synapses of the somatosensory afferent pathway (to initial cortical SEPs), which limits the capacity to reduce processing speed in this phase. The pathway from the site of stimulation to the contralateral parietal cortex, where the evoked potential was recorded, contains few synapses (located at the gracile nucleus in the medulla, the ventral posterior lateral nucleus in the cerebral cortex and terminating at the somatic sensory cortex) therefore limiting the ability to significantly reduce the N20 and P24-27 latencies. Previous evidence has demonstrated that more rapid reaction times could be generated when a stimulus can be sufficiently anticipated, by elevating the baseline level of activity integrator neurons, which initiates the response cascade [[31](#_ENREF_31)]. However, given the absence of differences in the N20 and P24-27 latencies between stimulus intensities, it is unlikely that participants were able to anticipate the impending stimulus in the present study. Notably, the similarity between the N20 and P24-27 waveforms and latencies indicates that the reduction of reaction time must occur further along the sensorimotor pathway at the level of cortical integration or during the efferent conduction to the flexor digitorum muscle.

## Conclusions

The current study set out to investigate the electrophysiological determinants (SEPs and ERPs) of rapid reaction times evoked by differences in stimulus intensity. We demonstrated that elevated stimulus intensity has no effect on the latency of SEPs while generating significantly shorter reaction and response times. Additionally, ERPs relative to the response time demonstrated a significantly greater pre-movement negativity to positivity following the high intensity stimulus, which may be related to movement planning and evocation of more rapid responses. This work has important implications for understanding the mechanisms by which the CNS processes the various characteristics of discrete stimuli that could be used to assist in novel rehabilitation methods for individuals who are characteristically slow to respond, such as individuals who have suffered a stroke. Further work is required to explore the potential role that emotional and attentional cortical centers may play to mediate the latency of responses when rapid responses are required.