ELECTROPHYSIOLOGICAL PATTERNS OF SKILLED MOTOR MOVEMENTS

by

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Skill implies a certain level of expertise when performing a voluntary movement. Research has identified that skilled movement is characterized by decreased cortical activation in the motor cortex and decreased coherence between neuronal populations. In a subset of the population, over-training can lead to the degradation of motor pathways of a specific skilled movement. Focal task specific dystonia, a movement disorder characterized by abnormal muscle contractions, can develop in people who are highly skilled at certain movements (musicians, athletes). Dystonic symptoms emerge when these patients are performing their well-learned movements. Previous EEG work has identified certain EEG signatures which are abnormal in dystonia. In this study, we investigated if similar beta frequency signatures and response patterns may be present in healthy people while performing familiar (‘skilled’) movements. We found that there is a significantly weaker beta power driven by post-movement beta rebound in ‘skilled’ trials compared to ‘unskilled’ trials, but no difference in pre-movement beta desynchronization or beta power during movement. Additionally, there is greater variability in response times during ‘unskilled’ trials, but no significant difference in incorrect responses. Our findings contribute to the field of skill-related research by examining ‘skilled’ conditions performed in healthy people as over-trained movements with the potential connection to focal task specific dystonia. Thus, our study suggests the need for further investigation in this relationship between skill-related and task-specific dystonic research.

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Introduction

While skill is usually associated with complex activities like playing an instrument or a sport, most everyone performs skilled tasks in their daily lives. These activities can include even the most mundane tasks, such as tying your shoes or jotting down notes. At one time, these tasks were foreign, but continued learning allows the motor control cortical pathways to adapt. As learning progresses, adaptive strategies develop and task irrelevant stimuli are suppressed. Processing of the environment and internally generated cues then become refined as the pattern becomes a skill (Fitts & Posner, 1967). Through consistent practice, a 'motor memory trace' is formed to facilitate task-related activation in the primary motor cortex. Whenever the task is presented again, the trace is efficiently 'retrieved' through activation but inhibited in all other contexts in healthy individuals (Hummel et al., 2002).

Like all pathways in the brain, motor memory traces are flexible and continue to change with more practice (Deeney et al., 2010). These pathways create more efficient attentional processing even as one approaches an expert skill level. Occasionally, in vulnerable individuals, activity in these pathways can go awry with over-practice. In some individuals, this can result in a movement disorder called focal task specific dystonia (FTSD). This disorder affects a once-refined movement and prevents the affected population from performing said movements without significant difficulty. (Herrojo Ruiz et al., 2008). Abnormal muscular contractions occur in the muscles associated with the over-practiced movement, but are only present when the affected person tries to perform the specific dexterous task.

Dystonia represents a diverse group of movement disorders. The primary symptoms are involuntary muscle contractions that cause abnormal repetitive movements. FTSD is a more specialized form of dystonia, only showing symptoms when a specific over-practiced fine motor skill is performed. FTSD causes highly localized loss of voluntary movement that only occurs in the musculature involved in the task (Furuya & Uehara et al., 2018). Notably, these symptoms are not triggered by other dexterous activities, suggesting that FTSD occurs through a neural malfunction that researchers in this field are mostly unfamiliar with.

FTSD was first described in the 19th century as physicians documented the development of writer's cramp in clerks. This dystonia commonly affects musicians, typists, painters, and athletes whose sport involves a fine motor skill, like golfers or marksmen. FTSD symptoms can begin to appear in individuals older than 30, with an average onset of age 40 in most vocations. In the general population, FTSD's prevalence ranges anywhere from 7 to 69 million affected individuals. However, when only high-risk groups are considered, FTSD's incidence is much higher. Studies show that 14% of patients seen at performing arts medical centers have FTSD (Stahl & Frucht, 2017). Without treatment, symptoms of FTSD endure indefinitely.

Dystonia can arise from an over-practiced task as demonstrated in animal models. Animal models of dystonia were successfully produced in monkeys by overtraining a fine motor movement. These monkey models developed the induced appearance of motor impairment issues similar to the cramps experienced by patients with FTSD (Quaratone & Hallet, 2013). The somatosensatory cortex of the monkeys with induced FTSD symptoms was less organized, with greater overlapping of

representations of individual digits in the cortex. Severe over-training may induce a change in the connectivity of sensory and motor cortices. Because dystonic symptoms can be induced in an animal model, it is plausible this same phenomenon could occur in humans with over-training.

Dystonic patients exhibit a lack of precise control over a specific voluntary movement. Therefore, focal task specific dystonia likely involves a neural malfunction and potentially the underlying structures involved in voluntary movement inhibition and/or activation. (Furuya & Uehara et al., 2018). Voluntary motor movements are complex processes that involve millions of neurons in the midbrain, forebrain, and beyond. Voluntary movements are initiated in the basal ganglia (BG), a collection of structures located in the deep cerebral hemisphere. One of the basal ganglia's main purposes is to control the motor cortex through both activation and inhibition. These interactions create a BG-cortical signaling loop. Through studying motor control disorders like PD and general dystonia in which the BG's normal functions are disturbed, neuroscience research is beginning to identify some of the neural circuits within the BG-cortical loop that function to produce movement (Herrojo Ruiz et al. 2008).

One hypothesis about the production of motor movements suggests that the movement begins with the activation of specific sensory structures within the basal ganglia by other regions of the cerebellar cortex. Once this signal is received and excites various neuronal groups in the basal ganglia, it can continue to the supplementary motor area (SMA) if not inhibited. In this neuronal loop, the basal ganglia act to channel information to the appropriate region of the motor cortex and

function as a filter for inappropriate movements. This loop displays both the activation and inhibitory functions of the basal ganglia. The movement is produced once the neuronal activity of the SMA reaches a certain threshold. Neurons within the primary motor cortex control the force and direction of the requested movement (Bear, 2016).

The activity of neuronal networks within the motor cortex can be measured by observing their electrical signals. When summed over large populations, these exhibit oscillatory activity, commonly referred to as brain waves. There are five classifications of neuronal-related band frequencies, spanning a range of frequencies from <1 Hz to >100 Hz. In motor control research, studies primarily deal with alpha, beta, and gamma band frequency waves. Alpha band rhythms (5-12 Hz) result from coherent activity in larger neuronal networks and are present in both the cortex and the thalamus (Lopes de Silva & Pfurtshceller, 1999). Beta (13-30 Hz) and gamma band rhythms (>30 Hz) are representative of locally restricted neuronal networks. These oscillations are present throughout the brain in both the cortex and subcortical areas. Generally speaking, beta and high gamma oscillations are thought to possess opposing roles in the motor cortex: beta inhibits, whereas gamma facilitates dynamic motor actions during preparation and performance (Seeber et al., 2016). Researchers discovered that beta frequency oscillations are prominent in sensorimotor activity over 50 years ago (Zaeppfel, 2013). There is significant data supporting the beta oscillation's relationship to motor activity and disruption in motor disorders. Our study therefore focuses on the fluctuations of beta band oscillations.

Beta frequency bands fluctuate in response to motor planning, execution, and input (Seeber et al., 2016). Oscillatory fluctuations reflect both the properties of

single neurons and the organization of these larger networks. Neuronal networks exhibit different states of synchrony in response to externally or internally paced events. Beta event-related synchronization (ERS) and event-related desynchronization (ERD) are the dominant signals that reflect the neuronal facilitation of movement (Lopes de Silva & Pfurtshceller, 1999).

When the sensorimotor complex is engaged, healthy individuals exhibit fluctuations in beta power. These can be motor responses to imperative stimuli, motor imagery, or self-paced motor movements. When a voluntary movement is planned, beta oscillations desynchronize and produce ERD electrical signatures. In this stage, there is increased excitability in neuronal populations. Multiple studies have confirmed that during movements, beta activity is suppressed in sensorimotor areas (Seeber et al. 2016; Neuper & Pfurtscheller, 2001). When voluntary movement is terminated, beta band activity resynchronizes quickly and the resultant ERS reflects an increase in neuronal network synchrony. Post-movement ERS is termed beta 'rebound' and is driven by short lasting bursts of beta power (Neuper & Pfurtscheller, 2001; Feingold, 2015). In short, movement planning and execution are characterized by beta band ERDs and movement termination is characterized by beta band ERSs. Beta ERDs are related to a specific event and show asynchronous neuronal activity through a reduction in beta power. Similarly, ERSs are increases in beta power that produce a synchronized electrical signal related to an event.

At the termination of movement, beta bursts are present and appear to be driving the resynchronization (post-movement ERS) of beta activity (Feingold et al., 2015). Bursts are defined as neuronal oscillatory activity periods above a certain power

threshold relative to the power level averaged across all trials. These bursts appear very close to the primary sensorimotor area that represent the used body part (Neuper & Pfurtscheller, 2001). In a finger tapping task conducted among healthy subjects by Jurkewicz et al. (2006), beta bursts occurred in the sensorimotor complex 230 +/- 170 ms after termination of movement and lasted for 680 +/- 170 ms. Thus, each beta rebound period is less than 1 s long on average, and many are required to drive ERS.

Each trial in our experiment is self-paced with a maximum time allotment of 3500ms and requires a maximum of four responses per trial. Thus, we expect to observe four separate sensorimotor beta (13-30 Hz) power decreases and rebounds in each trial corresponding to each movement in the sequence.

Neuroscientists use an electrophysiological monitoring device called an electroencephalogram (EEG) to collect oscillation data from the cortex. EEG can also be used clinically to monitor brain activity during sleep, diagnose epilepsy, and after a traumatic brain injury. EEG monitoring is a non-invasive procedure. An EEG 'cap' is secured on the head of the person under study that contains 64 detachable electrodes, each corresponding to an approximate location on the cortex. EEG recordings are notorious in neuroscience research for having poor spatial resolution. However, EEG has high temporal resolution, capturing electrical signal data within milliseconds. Activation and inhibition of neuronal networks can be detected by changes in EEG signals (Neuper & Pfurtscheller, 2001). Neuronal activity in certain frequency bands are expressed in two characteristic EEG patterns: amplitude attenuation (power decrease) and amplitude enhancement (power increase). In the context of the beta frequency, the amplitude attenuation (beta power decrease) observed in EEG data is synonymous to

beta ERD (Lopes de Silva & Pfurtshceller, 1999). Similarly, the amplitude enhancement (power increase) is synonymous to beta ERS, the post-movement synchronization.

Relevant Research Findings

Our study uses EEG to measure beta power fluctuations in response to external stimuli and under different trial conditions. In making our predictions, we focused on two fields of research that overlapped: skilled movement and FTSD studies. Studies performed on FTSD patients are relevant in examining the nature of skilled movements because they allow researchers to observe electrophysiological patterns associated with a disorder closely related to skilled movement. Therefore, contrasting the findings of dystonic studies with the findings of skilled movement studies can contribute to our overall understanding of skill level and expertise.

Skill level associated with an activity affects how the movement is perceived and how it is processed. The perceived degree of effort, beta activity, and automaticity are among some of the factors that change with skill level (Deeney et al., 2010). This experiment explores the differences in skilled and unskilled conditions in keyboard typing by examining behavioral and electrophysiological responses during trials consisting of ‘skilled’ and ‘unskilled’ movements. Skilled tasks are commonly executed in the context of an activity performed by a subset of the population so a comparison can be made between ‘novice’ and ‘skilled’ participants.

Deeney et al. (2010) performed a target-shooting experiment between ‘skilled’ and ‘novice’ marksmen. During the task, they examined coherence in alpha, beta, and gamma oscillations produced prior to, during, and after movement. In the low beta

frequency range (14-22 Hz), experts exhibited less overall coherence relative to novices at most every electrode pairing in the sensorimotor cortex. For the high beta frequency range, experts displayed decreased coherence in only the right hemisphere. Coherence refers to the degree of similarity between two EEG electrodes. If coherence is high, the two electrode signals have a very consistent phase and amplitude relationship and may indicate that these two areas of the brain are communicating (Fries, 2005). Because experts had pre-existing motor plans for the task, they exhibited less communication amongst neuronal networks in the motor cortex as different regions of the cortex did not have to work together to perform a more familiar task.

Hauffler et al. (2000) performed a study with similar parameters. Again, novice and expert marksmen were analyzed, but in this study the authors compared beta, alpha, and gamma frequency log –transformed EEG spectral estimates of each skill level. Within the beta band frequency range, the skilled marksmen exhibited less cortical activation, or beta power increase, at every examined region in both hemispheres with the exception of a single electrode over the temporal lobe (T3). These findings resonate Deeney et al.'s results, showing less coherence in expert marksmen and showing skilled tasks exhibit less beta oscillatory power.

Previous research suggests that individuals with more skill require less cortical activation. In this experiment, we will divide participants into relative 'expert' and 'novice' rankings based on their performance in a standardized typing assessment. We anticipate reduced fluctuations in sensorimotor beta power over all trial types for people with a higher skill level compared to those with a lower skill level.

In conjunction with studying skilled motor movements, we will be investigating how our data compares to existing knowledge of focal task specific dystonia. A common design of dystonic studies is to compare patients with FTSD and control subjects in a fine motor task. This can be the activity that triggers dystonic symptoms within the patient or an unrelated task. This experiment's task mimics the dystonic study structure without the use of an independent control group or a patient group. Instead, participants are presented with a keyboard task with two conditions. In one condition, the participant uses a familiar keyboard layout to respond to stimuli. In the other condition, the participant is asked to respond using an unfamiliar numbering scheme explained prior to the start of the task. We expect the familiar condition to exhibit 'skilled' behavior and electroencephalographic patterns. We want to find out if these familiar conditions will produce behavioral and encephalographic patterns similar to those found in dystonic patients.

As previously mentioned, it is possible to induce dystonic symptoms in animal models by over-training a specific dexterous movement. Because dystonic features can exist in models without an FTSD diagnosis, participants in this experiment may produce EEG signatures and behavioral results that resemble that of dystonic patients when performing very familiar movements.

Prior research has sought to determine the differences in functional connectivity between patients with focal dystonia and healthy subjects. In a finger tapping task sequenced task performed by Jin et al. (2011), patients with focal hand dystonia showed reduction of beta band functional connectivity in sensorimotor areas. This significant difference in beta functional connectivity existed in both the rest and task conditions.

According to Quaratone & Hallet's (2013) review of dystonic physiology, there are three physiological abnormalities that contribute to dystonic behavior. Besides altered synaptic plasticity and sensory dysfunction, the most significant physiological trait is the loss of surround inhibition. This reduction in ability to select necessary neuronal activity for motor output greatly contributes to the difficulty in focusing motor commands. This discovery resulted from activation and inhibition motor tasks performed on dystonic patients. Also termed Go/NoGo tasks, these prepare the subject for movement with a common cue between the two trial types, followed by a stimulus for activation or inhibition. Encephalographic analyses of beta power in these tasks have provided interesting insights to the specific dysfunctions in focal task specific dystonia. In healthy subjects performing a finger tapping task, Hummel et al. (2002) reported a notable increase in high alpha (11-13 Hz) oscillatory activity during inhibition trials and a decrease in this activity during activation (go) trials. In dystonic patients, there was no increase in alpha band activity during inhibition trials. During inhibition, EEG activity looked similar to activation trials across both the alpha and beta frequencies. Where an increase in alpha and beta synchronization was anticipated in healthy subjects, the synchronization was absent in the patients with dystonia.

In more complex motor tasks with activation and inhibition conditions, similar results are seen. In Herrojo Ruiz et al.'s (2008) study on piano players, musicians with task related dystonia and healthy pianists played piano scales when they observed the proper Go/NoGo cues. Rather than reporting an absence of synchronization, in this more complex task the synchronization was smaller and delayed for dystonic patients during inhibition trials. This lag in beta resynchronization had a latency of 850-900ms

specifically for the high beta (23-30 Hz) signals. This observation was significant for left premotor, left sensorimotor, and mesial frontal cortex (FC3, F3, FCz, Cz) areas for a task performed with the right hand.

In Go/NoGo studies performed with FTSD patients, error rates (i.e. activity in an inhibition trial) between control and patient groups were not significantly different (Ruiz et al, 2008; Hummel et al., 2002). However, times between responses of two subsequent notes were more variable in dystonic subjects. A later study performed by Furuya et al. (2015), analyzing the loss in dexterity in musician's dystonia, reproduced earlier findings and confirmed more rhythmic variability in dystonic patients compared to healthy musicians.

In the 'skilled' condition of our experiment, we predict similar results to what was observed in the activation trials of Go/NoGo tasks. Because dystonic models can be produced in animals through over-training of a skilled task, frequently practiced movements could have similar EEG signatures to that of dystonic patients. We expect reduced fluctuations in sensorimotor beta power (i.e. less of a decrease during movement a reduced subsequent "rebound" following movement) during 'skilled' compared to 'unskilled' movements. (Herrojo Ruiz et al. 2008, Jin et al. 2011; Hummel et al., 2002). Behavioral patterns of the 'skilled' condition could model some dystonic findings as well. We anticipate greater variability of response times during the skilled trials. This hypothesis is based on the observation that more variable response times were seen in dystonic patients when performing skilled (familiar) tasks (Furuya et al 2015; Herrojo Ruiz et al. 2008). We also anticipate that there will be no significant

difference in errors between skilled and unskilled trials (Herrojo Ruiz et al. 2008; Hummel et al., 2002).

Hypotheses

To summarize, there are five specific hypotheses we have formed to address this question of electrophysiological patterns in skilled movements and how they compare to FTSD EEG signatures. In regards to encephalographic analysis, we are investigating beta power fluctuations in the sensorimotor cortex and are pursuing three different analyses:

1) We expect reduced fluctuations in sensorimotor beta power (i.e. less of a decrease during movement and a reduced subsequent "rebound" following movement) during ‘skilled’ compared to ‘unskilled’ movements. (Herrojo Ruiz et al. 2008; Jin et al., 2001).
2) We anticipate reduced fluctuations in sensorimotor beta power over all trial types for people with a higher skilled level compared to those with a lower skill level. (Hauffler et al. 2000; Deeney et al., 2010).
3) We expect to observe four separate sensorimotor beta (13-30 Hz) power decreases and rebounds (corresponded to each movement in the sequence) within a trial for each character/number stimuli of the sequence. (Jurkewicz et al. 2006).

Additionally, we investigated response times and error rates of participant input to look for additional dystonic signatures:

4) We will see greater variability of response times during the skilled trials (Furuya 2015 & Herrojo Ruiz et al. 2008).

5) We anticipate there to be no significant difference in errors between skilled and unskilled trials (Herrojo Ruiz et al. 2008; Hummel et al. 2002).

Our findings will contribute to the field of skilled movement research by examining the potential relationship in skilled' and 'unskilled' parameters in highly similar activities.

Methods

Participants

Data was collected from 13 right-handed human subjects (n = 13, 9 female) aged 18-25 (mean age: 20.3) with no history of movement disorders, carpal tunnel, normal to corrected vision, no neurological conditions, and not currently on any psychiatric medications. History of carpal tunnel symptoms excluded participation because carpal tunnel can cause excessive wrist movements similar to hand dystonia (Drory, 1991) and it is likely this pre-existing condition would have caused discomfort during the experiment. Screening questionnaire collected demographic information and asked if participants use “touch typing” (i.e. resting all fingers on the home row and spacebar and returning to these keys when not in use). Recruitment was conducted through posters with contact information displayed around the University of Oregon campus and surrounding areas and with the University of Oregon Psychology Research Participant Pool (SONA). Subjects were compensated with a payment of $12/hr or, if scheduled through SONA, allotment of 1 credit/hr. The participants provided written informed consent in accordance with the institutional review board of the University of Oregon and Declaration of Helsinki.

Task Design

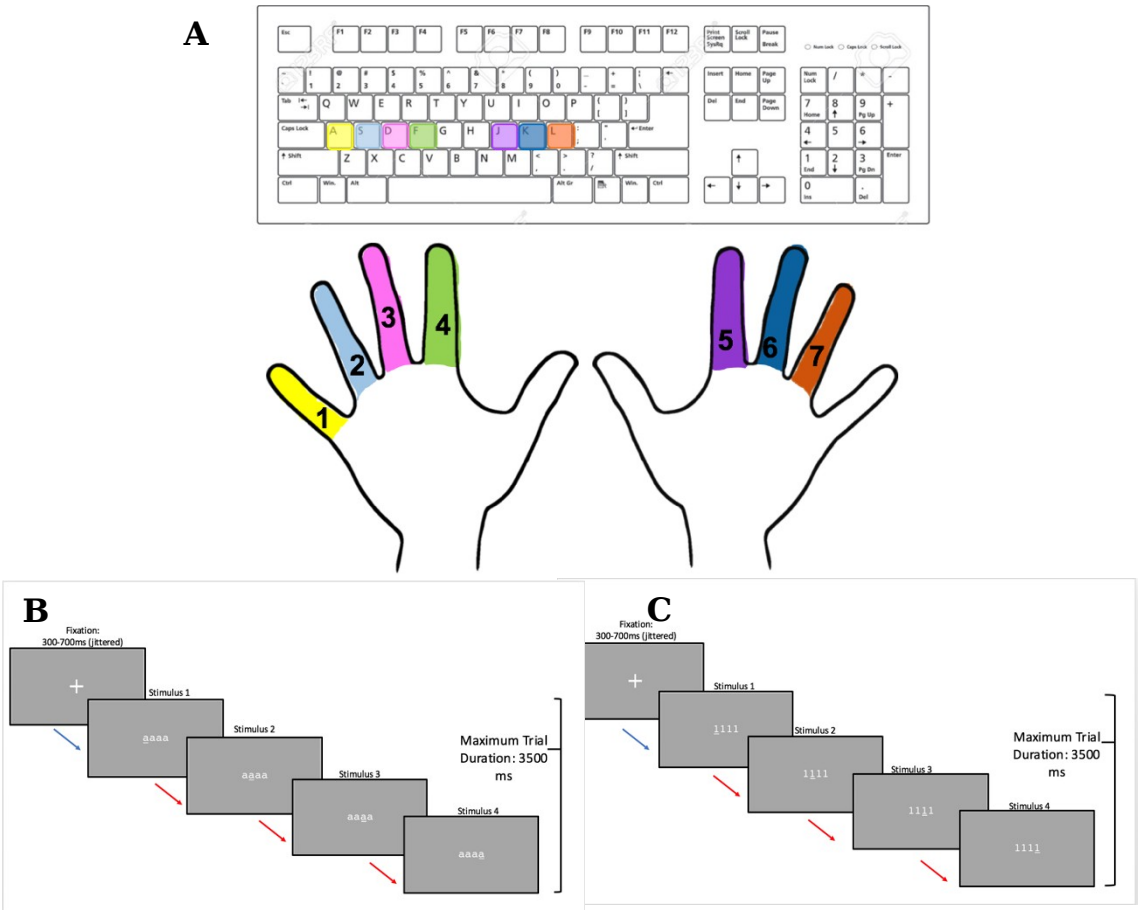


Figure 1: *Task Set-Up and Design*

(A) Response keys used in both 'skilled' and 'unskilled' trial types and finger numbering scheme for 'unskilled' trial types. Colors added to schematic only and were not present in the experimental set-up. (B) Progression of the 'skilled' trial type. Blue arrows indicate automatic advancement to next screen after jittered time segment, red arrows indicate advancement only if the participant responded with a key press. Total duration of trial was no longer than 3.5 s. (C) Progression of 'unskilled' trial, see (B) for details.

The behavioral task was presented to participants seated in a Faraday cage, an enclosure that blocks external electromagnetic fields, facing a computer monitor with a standard keyboard before them. Two trial types were tested: 'skilled 'and 'unskilled'. In the 'skilled' version of the task, a collection of four characters appeared on the screen.

There were seven potential characters the task could display: ‘a’, ‘s’, ’d’, ‘f’, ‘j’, ‘k’, or ‘l’. The selected characters corresponded to the keys on the home row of a standard keyboard with the exception of ‘g’ and ‘h’ because proper typing etiquette requires movement of the index fingers from their resting key to respond with these letters. Additionally, the ';:' key was excluded because behavioral piloting suggested that the inclusion of this key in a trial did not produce a natural response in the skilled trial types. Participants were instructed to respond to the ‘f’ character stimulus with their left index finger (L2), the ‘d’ character stimulus with their left middle finger (L3), and so on until their right index finger (R4) (Figure 1A). The right hand followed the same response pattern with the right index finger being responsible for the ‘j’ key and so forth with the exception of the right pinky finger. The thumbs (L1 & R1) and right pinky (R5) were not responsible for responding to any stimulus. In the ‘unskilled’ version of the task, a collection of four numbers appeared on the screen. The task displayed the first four entries of a randomized string of numbers 1-7. Each number represented one of the seven digits being used to respond to the trial stimuli. Participants fingers were numbered from left to right and excludes the thumbs (L1 & R1) and right pinky finger (R5) as the ‘skilled trial’ version did. The same keys participants used to respond to the ‘skilled’ version of the trial were used in the ‘unskilled’ version. The number ‘1’ asks the participant to respond with the ‘a’ key because it is beneath their left pinky finger (L5) (Figure 1A). The number ‘2’ refers to the key corresponding to their left ring finger (L4), and so on.

Prior to beginning the main task, participants performed a standardized typing assessment created by the research team. Each participant was allotted 60s to type as

many words as possible displayed on the screen before them. A research team member instructed the participant to begin and end typing when instructed and to focus on typing speed rather than accuracy. Each word was 4-6 characters long and displayed in a Courier font. Time was kept manually by the research team member and verbal start/stop cues were communicated to the participant. The text entry was displayed to the participant as it was entered in a Google Documents file. The calculated character per minute value was used in data analyses to examine relationships between typing skill and electrophysiological correlates.

To begin the experiment, participants read a series of instructions explaining how to respond to the 'letter' and 'number' trial types, which refer to the 'skilled' and 'unskilled' trial versions respectively. The experiment consisted of 12 blocks containing 40 trials each. The first two blocks and last two blocks contained only one trial type per block and were presented in a counterbalanced scheme, such that there were both a skilled and unskilled block at the beginning and the end of the experiment.

The eight middle blocks included both trial types in a randomized order. Each block contained 20 'unskilled' trials and 20 'skilled' trials. The order of the trials was randomized and participants were not aware of what trial type would be presented until the stimulus appeared and the trial began.

For each trial, a centralized white fixation cross appeared on a grey background prior to the stimulus onset. The fixation duration was jittered within a range of 300-700 ms with an experiment-based median of 500 ms with a uniform distribution. The character or number stimulus appeared immediately following the fixation cross. The character or letter combination remained onscreen for a maximum of 3.5 seconds or

when four responses were made (whichever comes first). A small rectangle appeared under the first stimuli and advanced to the next stimuli following each response (Figures 1B & C). There was a 1500-2000 ms period between the last stimulus presentation and presentation of the next fixation cross. Characters and numbers were presented in the monochromatic Courier font and set to a size of 50.

This behavioral task was created with MATLAB (2017a) using Psychophysics Toolbox Version 3 (PTB-3) functions.

Data Collection and Preprocessing

EEG data was collected using a 64-channel Active Two system sampled at 1024 Hz. Electrode offset was limited to +/- 20 mV. Eight additional electrodes were also used. These were placed as follows: two electrodes on the left and right mastoid as neutral channels, two on the left and right frontal zygomatic processes to monitor lateral eye movement, and two electrodes on each forearm approximately one inch apart and over the extensor digitorum to collect electromyography (EMG). EMG data was not analyzed at this time.

A photodiode was placed in the upper right hand corner of the participant's task presentation monitor to record changes in luminance which correspond to presentation of screen stimuli.

Prior to beginning the task, 3 minutes of resting EEG data was collected for each participant. At the end of the task, 3 minutes of resting EEG data was collected again.

Data preprocessing was performed using MATLAB 2017b and eeglab functions (A Delorme & S Makeig, 2004). To preprocess, the data from each electrode was re-referenced to average by first removing the mean of each channel and applying the

average reference. Once re-referenced, each dataset underwent high pass filtering at 0.5 Hz using a FIR1 filter to eliminate low frequency drift. All data underwent manual inspection to ensure quality. One dataset was excluded due to experimenter error in task presentation.

Independent component analysis was applied to the remaining data to separate data into maximally independent components. Components were manually inspected and ones corresponding to eye movement artifacts were removed. The data was then back-projected into channel space. Automatic rejection algorithms implemented in eeglab.m (pop_jointprob and pop_rejkurt) were then used to identify additional artifacts for removal.

Data Analysis

To address our hypotheses related to beta power differences between trial types, we generated spectrograms using MATLAB 2017b and eeglab functions. Per epoch, the power spectral density was calculated using the 'pwelch' function in MATLAB which utilizes Welch's method with a Hamming window per segment. The averaged log(power) from 13-30Hz per condition was used for statistical comparisons. More specifically, the change in log(power) between movement and non-movement periods were compared. Spectrogram plots decompose data into frequency ranges - highlighting the theta, alpha, and beta frequency power differences between 'skilled' and 'unskilled' trial types for the channels corresponding approximately to hand-area sensorimotor cortex (C3, C4). These were generated by filtering the data (using a FIR1 filter, eegfilt) between 2-100 Hz with a 3 Hz bandwidth. A Hilbert transform (Hilbert.m) was taken and the absolute value was extracted to derive amplitude over

time for each frequency range. Spectrograms were aligned either to the first stimulus onset in the trial or the individual keypresses. Furthermore, spectrograms of skilled trial type, unskilled trial type, and the amplitude differences between trial types were generated with each of the spectrograms listed above. Thus, for each trial condition, ten spectrograms were produced (five for each channel) to examine each point of reference. To normalize the data, baseline values were subtracted from the data. Alpha significance threshold values was set to $\alpha = .01$ to indicate differences with an uncorrected significance. However, no result between conditions survived correction for multiple comparisons.

To quantify beta power differences observed in points of interest in the spectrogram plots, we graphed box and whisker plots beta power differences corresponding to the first key press and fourth key press averaged across all trials for all subjects. Box and whisker plots were graphed for C3 and C4 channels for 'skilled' and 'unskilled' beta power differences between movement/post-movement rebound and non-movement periods. We sampled the 2700 ms- 1700ms period before movement (time = 0) to determine the non-moving parameters and 500-1500 ms after movement to determine the moving parameters for the first key press. We sampled 500ms - 1500ms after movement (time = 0) to determine the rebound parameters and 4500ms – 3500ms before the fourth key press to determine the reference the non-movement period before the start of the trial. The beta power difference values for the post-movement period were later compared to typing assessment scores to determine the correlation between skill level and beta power.

We performed an exploratory analysis on our data and graphed ERP plots for the five major events in each trial: the trial onset and four consecutive responses. Event related potentials were determined by filtering out for signals below 30 Hz using FIR1. Signals were aligned to events and averaged across subjects with the baseline removed. Additionally, we graphed additional points indicating times points where differences between ERP values in trial types were significant across all subjects. Significance threshold was set to $p<.05$.

To address our hypotheses related to reaction time variability, we averaged the first reaction times and the second, third, and fourth reaction times across all trials for all subjects. We excluded response times recorded as '0' because these denoted non-responses due to the trial timing out. We separated the first response time from the consecutive responses because the latency in the first response was greater amongst all subjects in comparison to consecutive responses. These were plotted in a box plot displaying the mean, 25^{th} percentile, 75^{th} percentile, and extremes. Additionally, statistical significance values were performed. Variance calculations are an unbiased estimate of the averaged variation times per subject and are not proportional to reaction times.

To address our hypothesis that response times related to error rates between trial types, we averaged all correct responses across all trials for each subject. Non-responses were included in the error calculations. The correct response percentage for each trial type was plotted in a box plot displaying the mean, 25^{th} percentile, 75^{th} percentile, and extremes. Additionally, statistical significance values were performed.

To create classes of ‘proficient’ and ‘novice’ typists, typing assessments were scored by total characters typed in the 60 second period and adjusted for errors made. Because participants were instructed to type as fast as they could without focusing on correcting mistakes, errors were calculated using the Total Error Rate equation that accounts for both incorrect fixed and incorrect not fixed entries (Arif & Stuerzlinger, 2009). Total error rate was subtracted from original characters per minute score for each participant to determine their net characters per minute score. The mean value of all collected assessments was set as the boundary to determine proficiency. With these qualifications, three subjects were considered ‘proficient’ and five subjects were considered ‘novice’. The first participant’s typing assessment data was excluded as we use their EEG data, and the entries of four additional participants were excluded because the files had been corrupted and therefore could not be accessed. Their EEG data was excluded from correlation analyses with typing data.

Results

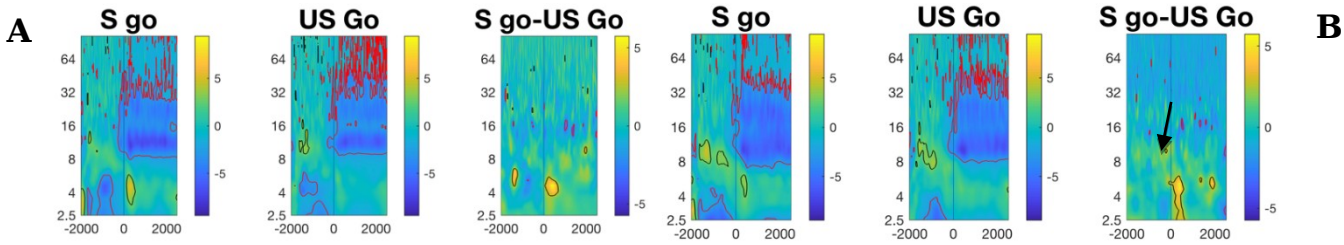


Figure 2: *Stimulus Cue and First Response Spectrograms*

(A)-(B) Spectrogram plots of power spectral density (x-axis = Hz, y-axis = ms since event). Statistically significant areas are outlined in red (for decreases in power) and black (increases in power) with a threshold of p = .01, uncorrected for multiple comparisons. Orange arrow indicates theta increase observed in skilled trials after go cue. S plots indicate skilled trial data, U plots indicate unskilled trial data, and S-U plots indicate trial differences. (A) Set to the trial start "Go" cue for C3 electrode channel. (B) Set to the trial start "Go" cue for the C4 electrode channel.

Electroencephalographic data was visualized using spectrogram plots for the five major events within each trial: onset of trial/go cue, first key press, second key press, third key press, and fourth key presses (Figures 2 & 3). Using these plots, we analyzed the spectral power fluctuations of each trial type independently and together by subtracting the difference. The first set of spectrograms (Figure 2), set to the trial start cue, display strong decreases in low beta (13-20 Hz) activity in both trial types and a weaker, but still significant, decrease in high beta (20-30 Hz) activity after the trial start cue, as expected (Pankelman, 2020). This is also apparent in the first key press spectrogram plots, as beta decrease initiates prior to the recorded key press and persists afterwards in preparation for the next response (Figures 3A&B). Beta decreases were present in both C3 and C4 electrodes. These plots did not display notable differences in beta activity between trial types. However, there is a significant and unexpected

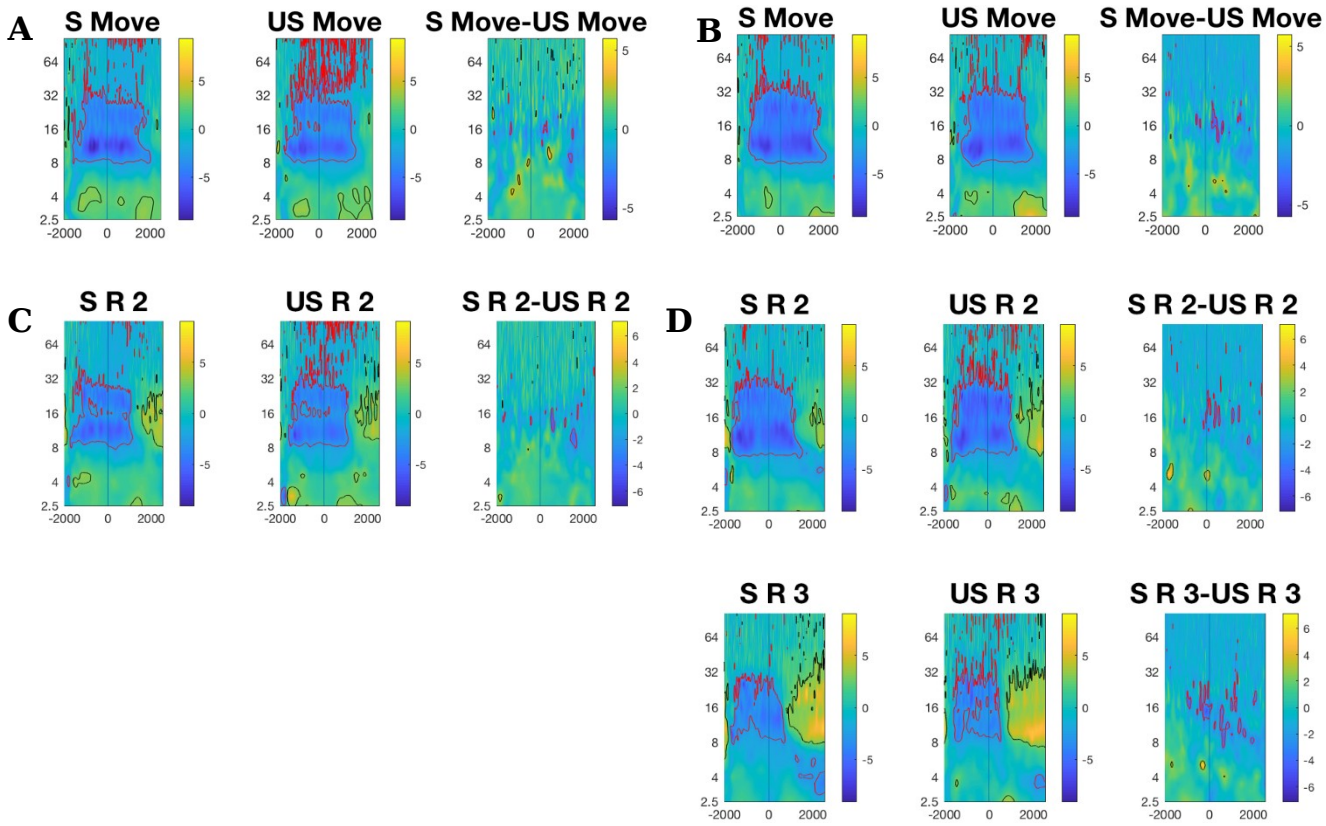
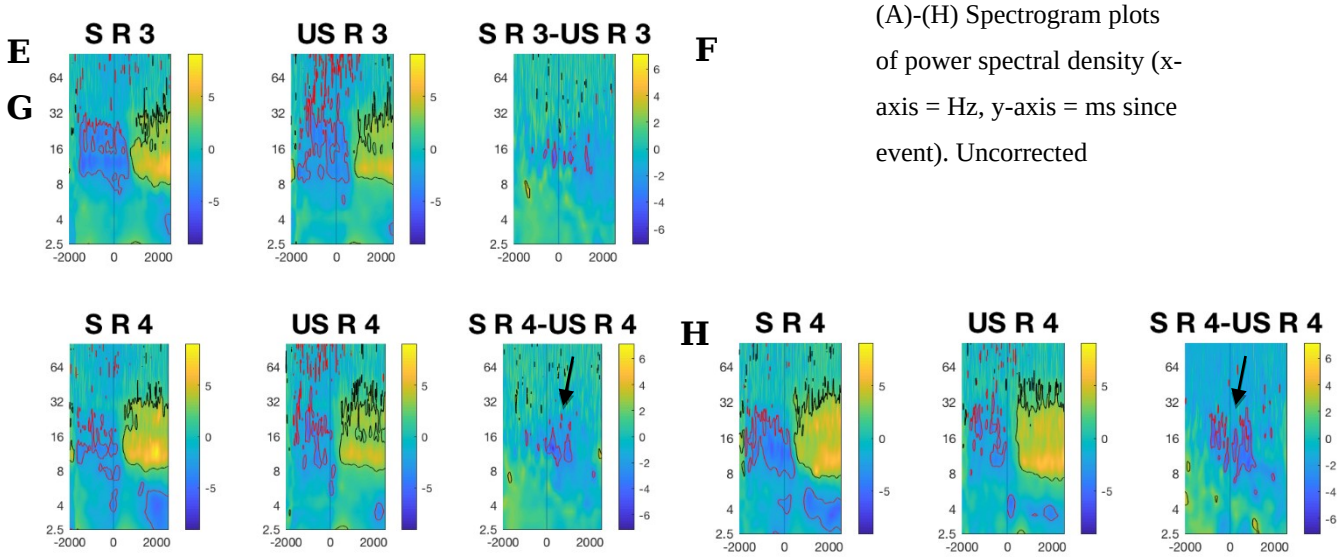

A
S Move
US Move
S Move-US Move
B
S Move
US Move
S Move-US Move
C
S R 2
US R 2
S R 2-US R 2
D
S R 2
US R 2
S R 2-US R 2
S R 3
US R 3
S R 3-US R 3

Figure 3: *Second, Third, and Fourth Response Spectrograms*



(A)-(H) Spectrogram plots of power spectral density (x-axis = Hz, y-axis = ms since event). Uncorrected statistically significant areas outlined in red and black with a threshold of p = .01. Black arrow indicates weaker beta rebound observed in skilled trials after fourth key press. S plots indicate skilled trial data, U plots indicate unskilled trial data, and S-U plots indicate trial differences. (A), (B) Set to the first recorded key press for electrodes C3 and C4 respectively. (C), (D) Set to the second recorded key press response for electrodes C3, C4 respectively. (E), (F) Set to the third recorded key press for electrodes C3, C4 respectively. (G), (H) Set to the fourth recorded key press for electrodes C3, C4 respectively.

difference in theta activity (3-7 Hz) between trial types on both sides of the motor cortex. Theta increases occurred was stronger in the skilled trial type immediately after trial start cue (Figure 2A&B).

There is no difference in beta desynchronization amplitude associated with ‘skilled’ trial types compared to ‘unskilled’, as displayed by the difference plots for each key press movement. The most notable finding displayed by the movement

spectrograms is the difference in beta rebound. There is a significantly weaker beta increase associated with the fourth key press in skilled trials (Figures 3G&H). This is present in both sides of the motor cortex, but is pronounced in the left hand (C4).

Beta power difference between movement and no movement states were plotted by trial type and channel for the first key response and the beta power increase following the last key response (Figure 4). In the first key press, beta power difference between movement and non-movement was not significant for the C3 or C4 electrode ($p_{C3, C4}$ = 0.5952, .2284). We sampled the 2700 ms- 1700ms period before movement (time = 0) to determine the non-moving parameters and 500-1500 ms after movement to determine the moving parameters for the first key press. In the time after the fourth key press, beta power difference was significant between rebound and non-movement states for right handed movement (C3), but not left handed movement (C4) ($p_{C3, C4}$ = 0.0297, .1155). We sampled 500ms - 1500ms after movement (time = 0) to determine the rebound parameters and 4500ms – 3500ms before the fourth key press to determine the reference the non-movement period before the start of the trial.

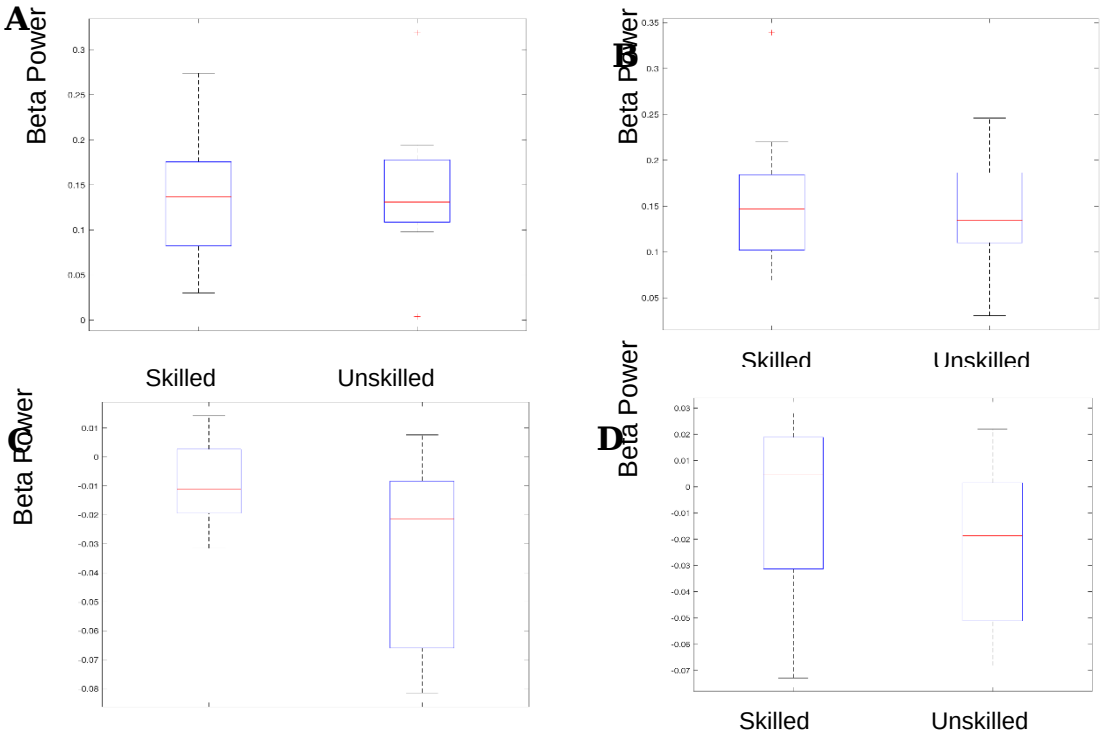

A
Beta Power
B
Beta Power
Skilled
Unskilled
C
Beta Power
D
Beta Power
Skilled
Unskilled

Figure 4: *Beta Power Differences by Trial Type*

(A)-(D) Beta power difference of movement/no-movement (A)-(B) and rebound/non-movement (C)-(D) states averaged across subjects separated by trial and response types. Center red line indicates median, box edges are 25th and 75th percentile, whiskers extend to most extreme data points, and red crosses represent outliers (more than three standard deviations from the mean). (A), (B) First key press response for all skilled and unskilled trials for C3, C4 respectively. (C), (D) Beta increase associated with fourth key press response for skilled and unskilled trials for C3, C4 respectively.

Event related potential plots for the five major events per trial showed multiple significant time points across all five events (Figure 5). ERP plots with the highest number and greatest density of notable time points correspond to the go cue and the fourth key press (Figure 5A & 5E). In the plot aligned to the go cue, there is a cluster of significant data points immediately following the presentation of the go cue (time = 0). In the plot aligned to the fourth key press, there is another cluster of significant data points immediately after movement has terminated for the entire trial. The ERP plot aligned to the third key press for channel C4 (Figure 5C) present a smaller, but still notable, cluster of significant data points. The significant data points did not survive a multiple comparisons test.

Skilled Unskilled

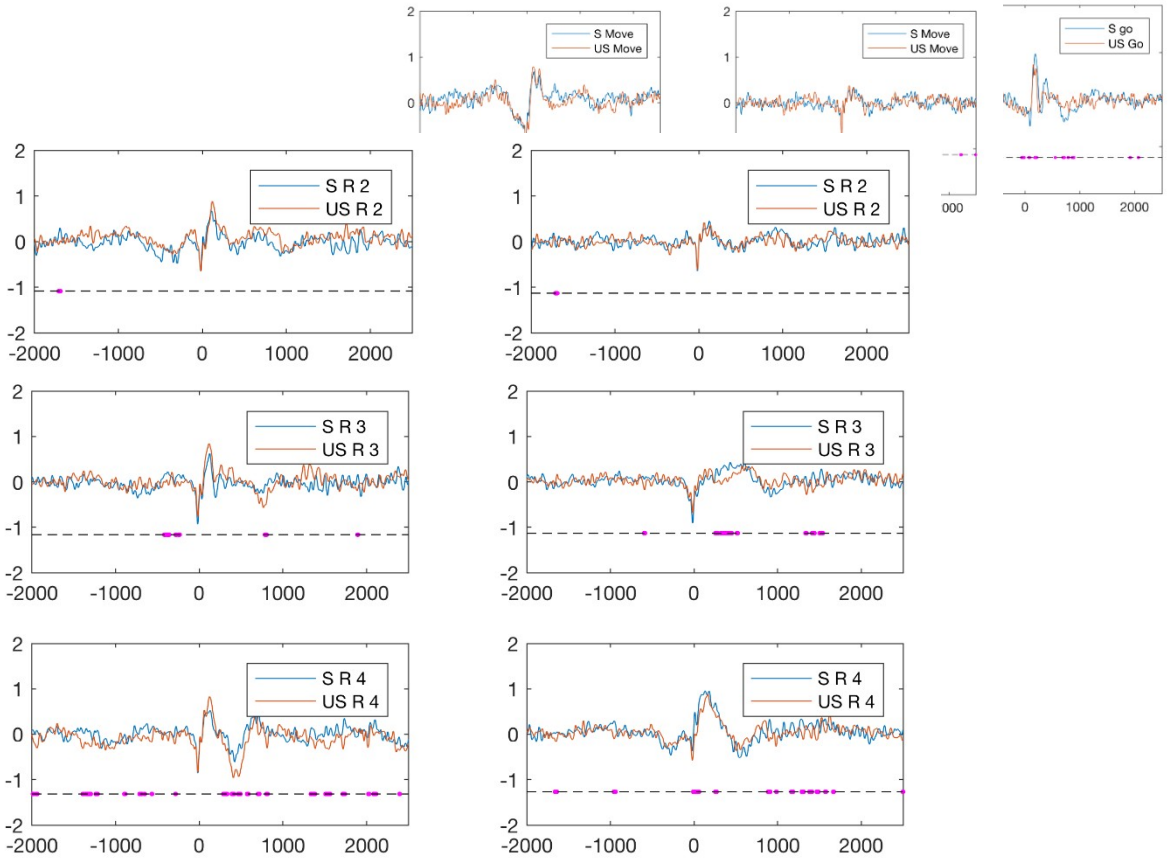
To analyze how beta power during movement and non-movement phases differed with participant skill level, we determined proficiency across subjects and correlated these values with beta power differences in the post-movement rebound. To determine proficiency, errors were subtracted from total characters typed and scores

were averaged across all subjects with available assessment data (mean = 155.87, n=8). Because there was a significant difference in beta rebound between the fourth responses in skilled and unskilled trials, scatter plots (Figure 6) are shown for beta power differences in the post-movement rebound in both trial types. No significant correlations exist in beta power in either trial type or channel. Additionally, we examined the correlation beta power differences in the first response and typing proficiency, but these comparisons showed no significant correlations either.

Our behavioral analysis examined variability in response times and percent of correct responses by trial type. First response times were separated from consequent responses because of the increased latency in the first response across all subjects (mean $_{\text{R1 skilled}}$ = 1.1030; mean $_{\text{R1 unskilled}}$ =1.0800 as compared to mean $_{\text{R2-4 skilled}}$=.3573, mean $_{\text{R2-4 unskilled}}$= .4085). We found that variability for the first response for each trial was not significantly different (p =.9194), but was significant in other responses as unskilled key

presses showed a greater variability later in the trial (p=.0086) (Figure 7A & 7B). Meanwhile, the percent correct responses across all key presses were not significantly different in each trial condition (p= .0810; mean error $_{skilled}$ =85.46%, mean error $_{unskilled}$ = 90.15%) (Figure 7C).

Figure 5: *Event Related Potential for Skilled and Unskilled Trials and Different Trial Events (legend on next page)*



(A)-(B) Event Related Potential (ERP) Plots for skilled and unskilled trial types set to different trial events. S indicated skilled trial data and is graphed in blue, U indicates unskilled trial data and is graphed in orange. X-axis values are time in ms, y-axis values are potential in μV. Significant time points ($p<.05$) are indicated with magenta markers on black dashed line. (A) ERP Plots for skilled and unskilled trial types set to trial start "Go" cues in the top two graphs. (B) ERP Plots for skilled and unskilled trial types set to first response (Move), second response (R2), third response, (R3), and fourth response (R4).

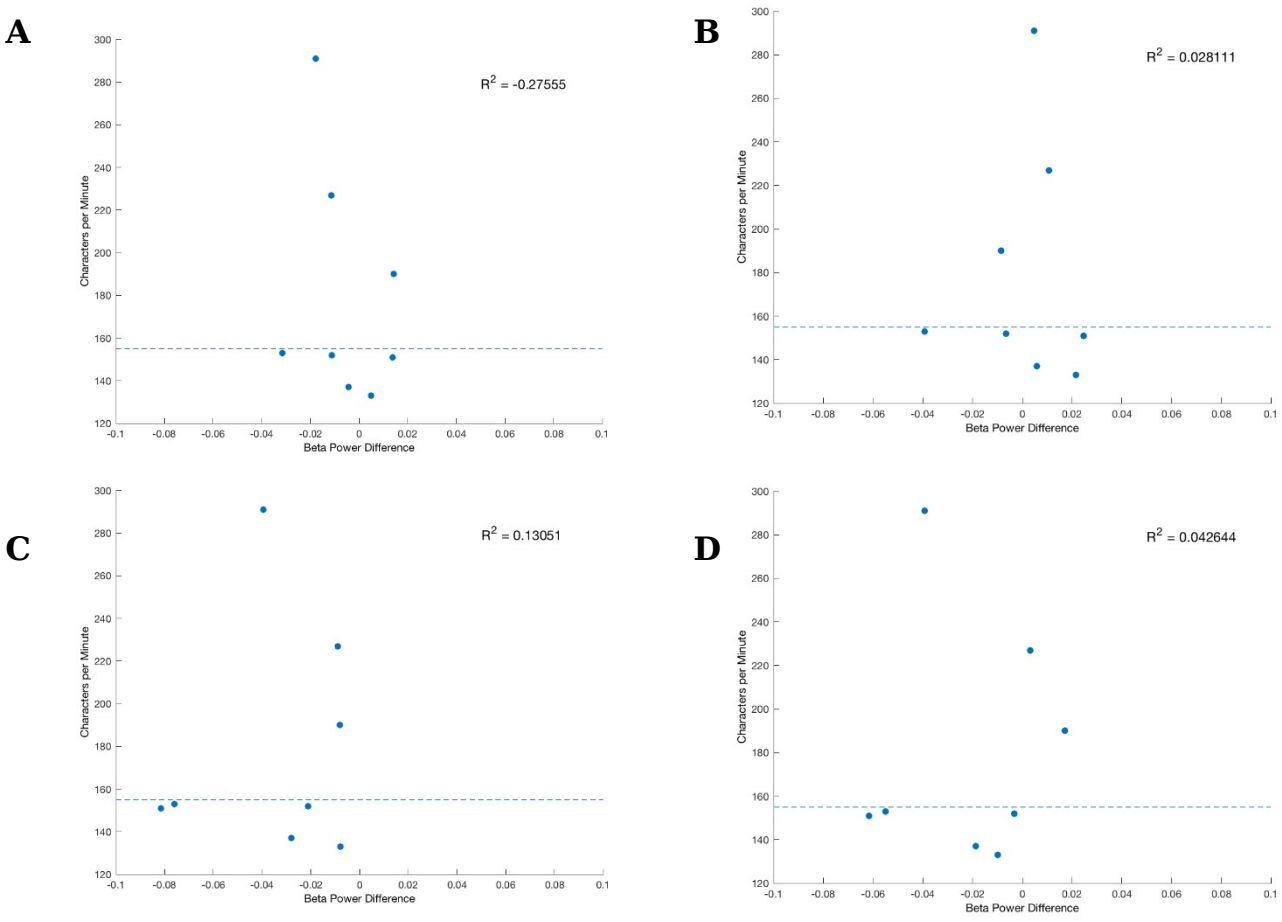


Figure 6: *Beta Power and Typing Proficiency Correlations (legend on next page)*

(A)-(D) Scatter plots of typing assessment data in characters per minute (y-axis) and beta power rebound/non-movement log(power) (x-axis). Dashed blue line indicates the typing assessment score that qualified participants as 'novice' or 'proficient'. Scores above the dashed line considered proficient typists based on our assessment. R^2 values displayed in each graph. (A), (B) Beta power difference in skilled fourth responses in channels C3, C4 respectively. (C), (D) Beta power differences in unskilled fourth responses in channels C3, C4 respectively.

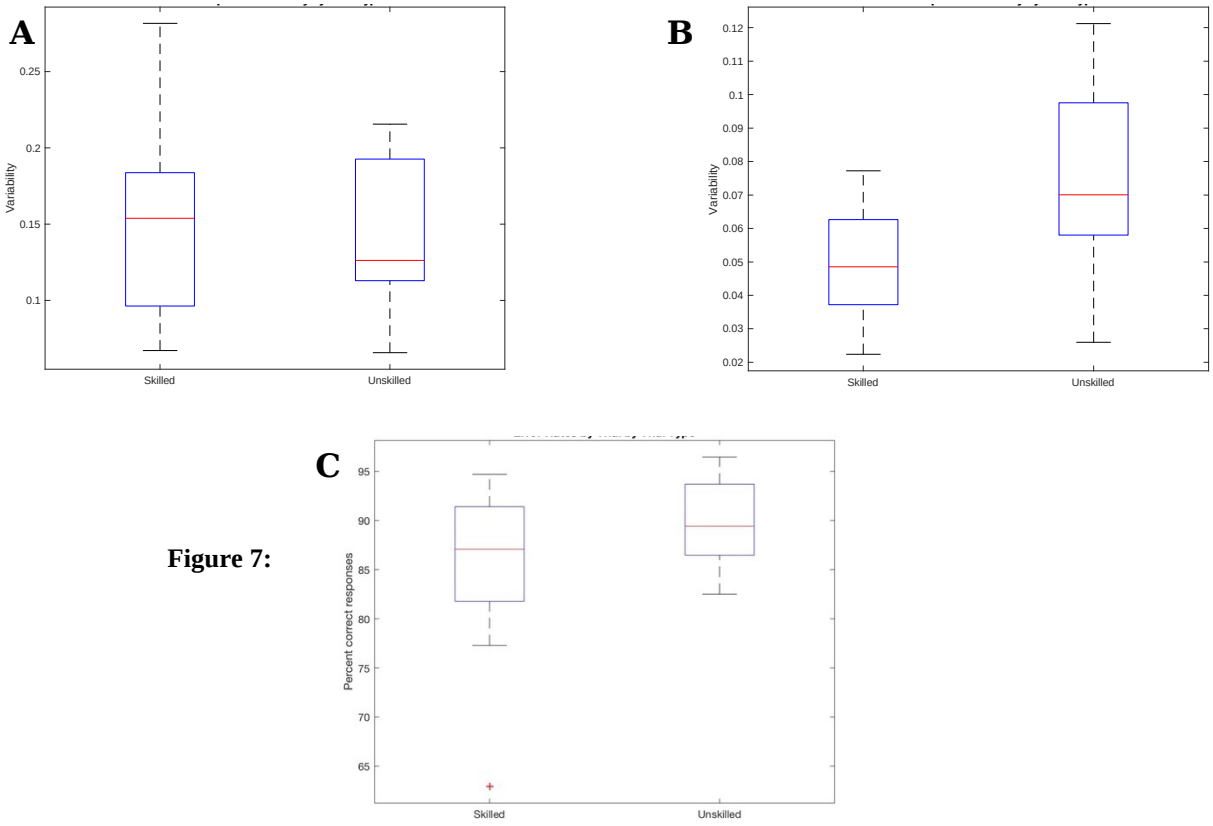


Figure 7:

Errors and Response Time Variability

(A) Box and whisker plot of response time variability for first keypresses in skilled and unskilled trial types averaged across subjects. Center red line indicates median, box edges are 25th and 75th percentile, whiskers extend to most extreme data points, and red crosses represent outliers (more than three standard deviations from the mean). (B) Box and whisker plot of response time variability for second, third, and fourth key presses for skilled and unskilled trial types. (C) Percent errors for skilled and unskilled trial types averaged across all subjects. Error calculations included non-responses while response time variability did not.

Discussion

In this study, we sought to compare EEG signatures of healthy individuals performing two very similar activities under ‘skilled’ and ‘unskilled’ conditions. We conducted this investigation by testing five hypotheses related to sensorimotor beta power modulation and behavioral data.

We expected to observe reduced fluctuations in sensorimotor beta power during ‘skilled’ trials compared to ‘unskilled’ trials. We were anticipating a decrease in beta desynchronization onset by the trial start cue and a weaker beta rebound following movement offset. Our results partially agreed with this hypothesis. There was no significant difference in beta desynchronization between trial types (Figures 2 & 3A-B). In accordance with our predictions, we saw decreased beta power driven by a weaker beta rebound after the fourth key press in ‘skilled’ trial types compared to ‘unskilled’ (Figure 3G & 3H). This decrease in ‘skilled’ beta power is consistent with EEG signatures observed in patients with FTSD (Herrojo Ruiz et al., 2008). Additionally, we observed an unexpected increase in the theta band frequency activity during the ‘skilled’ trial types immediately following the trial onset cue (Figure 2). We speculate at this stage that the increase in theta activity after observing the character stimuli may reflect a variation of ‘response conflict’. Response conflict occurs when interference of irrelevant stimulus affects a participant’s reaction time to the relevant stimulus (APA, 2020). Because the ‘skilled’ trial type presented letters in a random order, participants may have experienced response conflict as they saw characters, but no words, and had to suppress irrelevant information that may have arisen from this conflict. Various studies have shown that conflict leads to an increase in theta power and hypothesized

these originate in the medial frontal cortex (MFC) (Nigbur et al. 2012). Although the channels we investigated primarily observe signals from the sensorimotor cortex, there is significant volume conduction in EEG, such that MFC signals may have registered in the C3 and C4 electrodes. It is important to note that we did not see a difference in first response time latencies between trial types. Although theta frequency activity was not our first interest, this could be worth further investigating with more subjects and analyses focused on theta activity and how response times to character strings may differ from complete words.

Our hypothesis examining reduced beta frequency fluctuations provided the strongest connection of this study to electrophysiological results found in dystonic literature. As previously stated, the primary purpose of this study was to compare the EEG signatures of healthy subjects performing familiar tasks to EEG signatures of FTSD patients. Because of results published in dystonic papers, we expected to see weaker beta desynchronization and weaker beta rebound driven synchronization when participants performed skilled trials (Herrojo Ruiz et al. 2008; Jin et al., 2001). We did not observe dystonic signatures during the ‘skilled’ trial type beta desynchronization period, but we did observe a smaller beta increase potentially driven by beta rebound in these trials. It is possible we were under-powered to observe a difference in beta desynchronization. This could also be the result of the nature of beta desynchronizations. Recent literature investigating these beta signatures has shown that post-movement beta rebound is much more responsive to changes in activity parameters than the pre-movement beta desynchronization or the movement-related beta decrease

(Pakenham et al., 2020). Regardless, our experiment shows patterns similar to dystonic signatures, but more subjects are needed before we can assert these claims.

In addition to the spectrogram plots, we also plotted beta power differences between movement and non-movement time points in reference to the first key press (Figure 5A & 5B) and the beta power increase following the fourth key press to non-movement (Figure 5C & 5D). Statistical comparisons revealed that these beta power comparisons yielded no significant results in both channels for first response analysis and in C4 for the post-movement beta increase. The difference between 'skilled' and 'unskilled' beta power increase was significant for the beta rebound period in channel C3 ($p=.0186$). For the beta decrease following the fourth keypress, we sampled the 500ms to 1500ms after and 45000ms to 35000ms before the fourth keypress to determine the rebound driven beta increase and non-movement parameters respectively for the fourth key press. Higher skilled values indicate less of a beta rebound because non-movement beta power was subtracted from the rebound power. Thus, our beta power analyses coincide with the weaker beta increase we observed in 'skilled' trials. However, this is only true for the C3 channel. In our spectrogram plots, significance was observed in both sides of the sensorimotor cortex. This could stem from an issue with the defined non-movement and rebound times. Further analysis is needed to determine the best time parameters before establishing the beta power differences in only a single channel as significant.

Based on previous literature analyzing beta rebound latencies, we hypothesized that we would observe four separate beta power decreases and rebounds within a trial for each of the stimuli presented. Our hypothesis was formed in part because we

anticipated that the participants would process each stimulus semi-individually due to their discrete nature. Additionally, previous literature cited beta rebounds lasting less than 1 second on average (Jurkewicz et al., 2006), and predicted that in a 3500 ms trial, multiple beta rebounds could occur. However, our results more closely resembled a continuous reduction in beta power throughout the trial following the initial desynchronization from the trial start cue (Figure 1 & 2). This decrease in beta power was consistent for both C3 and C4 electrodes as well as trial types. This may infer that participants did not observe each stimulus individually, but rather observed the string of stimuli prior to making their first response, and perhaps programmed the 4 button presses as a single motor command. The increased latency of the first response times support this theory. Consecutive responses may have been much quicker because all stimulus processing was performed at the beginning of the trial.

Our final EEG analysis compared sensorimotor beta power fluctuations to participant typing proficiency. Because post-movement beta power rebound produced significant results in the spectrograms, we primarily used beta power data from the beta increase following the fourth key press (Figure 4C & 4D) to compare to participant typing score. Beta power data from the first response was also compared to typing scores to check for possible correlation, but these results were statistically insignificant. There were no significant correlations observed in skilled or unskilled beta power values and typing ability in the C3 or C4 electrode (Figure 6). Similar results have been reported in skilled literature. Vogt et al. (2017) examined readiness potentials (RPs) and EMG in skilled novice, unskilled novice, and expert archers. Readiness potentials refer to the slow negative descents in EEG potential preceding movements and share some

features with beta desynchronization. The authors found no significant difference in RP's of motor areas when comparing skilled and unskilled novices, much like the beta power comparisons of this study. Because our analysis was limited to eight participants and some statistic tests could not be performed with samples of this size, more data must be collected before claiming there is no correlation between typing proficiency and beta power.

In addition to spectrogram and beta power plots, we performed an additional exploratory investigation and determined ERPs for five significant events in each trial: the trial start cue and the consecutive four key presses (Figure 5). Time points where 'skilled' and 'unskilled' potentials were significantly different ($p < .05$) were graphed within each plot. The ERP plots set to the trial start cue and fourth response contained the greatest number of significant data points. These results may resonate our spectrogram plot findings as there is a significant difference in electrical activity immediately following the trial start and the termination of responses. However, the ERP plots cannot provide frequency information, so based on these plots alone we cannot say these are driven by changes in theta or beta power. Taken together with our findings from our spectrogram plots, this provides stronger evidence that there were significant theta increases and beta decreases in the skilled trials in addition to confirming the accuracy of our time locking to stimuli in all other analyses.

We also sought to investigate differences behavioral responses between the two trial types. We were interested in comparing the results of 'skilled' and 'unskilled' trial types to results of healthy and dystonic patients. Dystonic literature reported that patients with FTSD did not have significantly different error rates compared to healthy

individuals (Herrojo Ruiz et al. 2008; Hummel et al. 2002), but did have greater variability in response times (Furuya 2015 & Herrojo Ruiz et al. 2008). In our experiment, there was no significant difference in error rates between trial types (Figure 7). This result is consistent with dystonic literature. To address the variability between response times, we divided the first response and later responses due to the increased latency with the first key press across all subjects. The variability in the first key press times showed no significant difference. However, the variability between second, third, and fourth responses were significantly greater during the 'unskilled' trial types. The variability in later responses is not consistent with FTSD patient results (Herrojo Ruiz et al., 2008), but does confer with data from previous skill-associated behavioral results. In one such study, Deeney et al. (2010) found the variability between trigger pulls to be significantly greater within the novice marksmen group. This difference in variability between first and consecutive responses is unexpected and to our knowledge has not yet been explored in dystonic or skill-related literature, likely because it is unusual to analyze the first response and consecutive responses separately.

Limitations

It should be noted that although we completed several statistical analyses for the EEG data, we did not perform corrections for multiple comparisons. A challenge to conventional multiple comparison corrections is that we assume independence of tests, which is not the case for EEG data where sample points, frequencies, and electrodes are dependent by design. A future direction of this work would be to perform a cluster-based correction (Maris & Oostenvald, 2007).

The task design presented limitations in its effectiveness to mimic unfamiliarity in the ‘unskilled’ task design. Participants who had learned to play an instrument using a hand numbering sequence may have had an advantage in the ‘unskilled’ trial type and thus found this to be more similar to a ‘skilled’ assessment. This conflict in task design could be alleviated by performing a similar study with individuals who use almost exclusively use a keyboard interface in their occupations (secretaries, stenographers, etc.). The potential familiarity of the ‘unskilled’ task might still prevail, but in comparison to their typing proficiency it would have an unskilled effect.

Another potential complication in the task design is the unavoidable training that occurs with the ‘unskilled’ stimulus response. Subjects completed 240 trials using the finger-numbering scheme in the ‘unskilled’ trial type. Acquaintance with the task likely resulted in behavioral and EEG patterns that appeared more similar to ‘skilled’ trial type data as the experiment progressed. Analyzing both behavioral and EEG data from ‘unskilled’ trials that occurred earlier in the experiment may produce different results that eliminate or reduce the influence of task acquaintance from the data.

Importance of Work

The findings of this paper may have implications for both skill-associated research and focal task dystonia research. Prior skilled-movement literature has mainly assessed the conditions of ‘skilled’ and ‘unskilled’ tasks in separate subjects. This study takes on a new approach of emulating these conditions in a single subject and observing how the conditions change their behavioral patterns and sensorimotor beta activity. More importantly, this study’s results could have clinical implications because of its

connection to the FTSD movement disorder. Identifying dystonic signatures in healthy people could assist in the creation of an early diagnosis test for at-risk FTSD groups.

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