THE EFFECTS OF VIOLENT VIDEO GAME ADDICTION ON WORKING MEMORY: AN ERP STUDY

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THE EFFECTS OF VIOLENT VIDEO GAME ADDICTION ON WORKING MEMORY: AN ERP STUDY

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ABSTRACT

THE EFFECTS OF VIOLENT VIDEO GAME ADDICTION ON WORKING MEMORY: AN ERP STUDY

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The effects of video games on cognitive processes are still controversial. Previous studies revealed enhancement in working memory performances of violent video game players, whereas some others showed negative effects on working memory performance. The current study aims to investigate the effect of violent video game addiction on working memory performance by using event-related potentials (ERPs). Total of 40 (20 addicted, 20 non-players) university students (18-31year-old) participated to the study. We hypothesized that event-related potential (ERP) responses of violent video game addicts under working memory task would be different from non- players'. In light of this hypothesis, an adapted version of working memory task by Harkin and Kessler (2009) was used, and behavioral data and ERPs of participants were recorded during different phases of working memory task. Working memory task consisted of four phases: encoding the identity and

locations of the letters, indicating of the location of a specific letter (probe-1), indicating if the given letter was correctly located with respect to the originally encoded set (probe-2), and degree of confidence for the responses of third phase. Stimulus presentation, recording, storage, and analysis were carried out via a 32 channel EEG/EP NeuroScan system. Behavioral results showed that the addicted group was faster than control group for incorrect responses in probe-2 phase. ERP results demonstrated that working memory performance were associated with the components recorded at at 100, 200, 300 and 400 ms time window, namely- N100, N400, P200 and P300. Detailed analyses were carried out for frontal electrode regions. Results indicated that working memory performance was demonstrated decreased amplitude and increased latency of P300 in addicted group, especially when cognitive load increased. Our findings supported previous neuroimaging studies that suggested decreasing amplitudes and increasing latency of P300 component is associated with violent video game addiction. Reduced P300 amplitude and increased P300 latency were detected also in individuals with substance dependency. Since P300 was associated with evaluative decision-making, divided attention and attention allocation, violent video game addicted group elicit decreased P300 amplitude which indicated worse performances.

Keywords: Working memory, violent video game addiction, event-related potentials

ÖZ

ŞİDDET İÇERİKLİ OYUN BAĞIMLILIĞININ ÇALIŞMA BELLEĞİ ÜZERİNDEKİ ETKİSİ: OLAY-İLİŞKİLİ POTANSİYEL ÇALIŞMASI

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Video oyunlarının bilişsel süreçler üzerindeki etkisi hala tartışmalı durumdadır. Önceki çalışmalardan bazıları şiddet içerikli oyun oynayan kişilerin çalışma belleği performanslarında gelişme, bazıları da azalma olduğunu ortaya koymuştur. Sunulan çalışmanın amacı şiddet içerikli video oyun bağımlılığının, çalışma belleği performansı üzerindeki etkisini olay ilişkili potansiyeller (OİP) yoluyla incelemektir. Şiddet içerikli video oyun bağımlılarının çalışma belleği görevi altındaki OİP tepkilerinin genlik ve latans değerlerinin oyun oynamayan katılımcılara kıyasla farklı olacağı hipotez edilmiştir. Çalışmaya 18-31 yaş aralığında toplam 40 (20 oyun bağımlısı, 20 oyun oynamayan) üniversite öğrencisi katılmıştır. Bu hipotezden hareketle, Harkin ve Kessler'in (2009) çalışmasında kullandığı görevden uyarlanarak yeniden oluşturulan çalışma belleği görevi kullanılmış, katılımcıların davranışsal verileri ve OİP yanıtları görevin farklı aşamaları süresince

kaydedilmiştir. Çalışma belleği görevi 4 aşamadan oluşmaktadır: ekranda çıkan harfleri ve yerlerini (içinde yer aldıkları kutuyu) öğrenme, verilen harfin yerini hatırlayarak gösterme (probe-1), verilen bu harfin yerinin doğru olup olmadığını belirtme (probe-2) ve 3. aşamadaki yanıtların doğruluğundan eminlik derecesini belirtme. Uyarıcı sunumu, kayıt, depolama ve analiz 32 kanal EEG / EP NeuroScan sistemi kullanılarak gerçekleştirilmiştir.

Davranışsal sonuçlar, harfin yerini doğru hatırlama aşamasında bağımlı grubun harfin yeri yanlışken (çeldirici kullanıldığınd) yanıt vermede kontrol grubundan daha hızlı olduğunu ortaya koymuştur. Çalışma belleği görevi sırasında N100, P200, P300 ve N400 zirveleri elde edilmiştir. Frontal bölgeden alınan kayıtlar üzerinden yapılan detaylı analiz sonuçları, görevin bilişsel yükü arttığında şiddet içerikli oyun bağımlılarının P300 genlik değerlerinde küçülme ve latans değerlerinde ise gecikme meydana geldiğini göstermiştir. Bu açıdan, çalışmanın bulguları, bağımlılık ve P300 ilişkisini inceleyen alan yazınını destekler niteliktedir. İlgili alan yazında genlikteki bu küçülmenin değerlendirerek karar verme ve dikkatin bölüştürmesi/ dağıtılması ile ilgili bir yetersizlik olabileceği vurgulanmış ve aynı özellik, alkol, madde ve sigara gibi bağımlı gruplarla yapılan çalışmaların bulgularıyla da benzerlik göstermiştir.

Anahtar Sözcükler: Çalışma belleği, şiddet içerikli video oyun bağımlılığı, olay-ilişkili potansiyeller

To my dear uncle who always watches us from the stars….

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CHAPTER 1

INTRODUCTION

Video games became a part of mass culture, after their invention in the 1980s (McDermott et al., 2014). Since it has been easier to access computers and game industry has rapidly been growing, excessive video game playing increased (Bartholow et al., 2006). Playing on-line video games (mostly violent ones) via computers, game consoles (e.g., Nintendo Wii, Sony PlayStation), or handheld devices (cellular phones, tablets) are among the most popular and entertaining leisure activities not just for children and adolescents but also for adults (Kirsch et al. 2005; Weber et al., 2006; Regenbogen et al., 2010). With technological advances, quality of video games, computer graphics and sound technologies has increased (Bartholow, Sestir & Davis, 2005), and new forms of video game playing showed up. Indeed, people can play with other people on online game platforms (Elmore, 2012; Kuss, 2013).

Playing online video game gives economic opportunities for players, too. Game players earn money by online broadcasting via their YouTube channel during playing a game video, via international contests on online game platforms (especially in countries such as Korea and Japan) (Kuss, 2013), and by selling their game avatars

(Lim & Reeves, 2009). Since playing games become excessive due to providing psychological and economic opportunities, questions have increased about whether it entails a risk or it is beneficial for well-being of people. Thus, researchers have started to investigate individual, cognitive, neurobiological, and social outcomes of excessive video game playing. According to Littel et al. (2012), it might be useful to determine the motivating factors of excessive game playing in order to identify individuals at risk and to have a better understanding of the behavior.

One of the main concerns against video games is that most of them are claimed to include violent elements (Griffiths, 1999). Another concern is that playing video games could have a positive or negative impact on core elements of cognitive abilities such as attention, concentration, reaction time, visual tracking, memory, mathematical ability, and verbal ability (Kuss, 2013). In this study, working memory performances of game addicts was compared with non-players by using Event-Related Potentials (ERPs) methodologies.

1. 1. Game Addiction

The term addiction refers to not only substances such as alcohol or drugs but also many constructs including internet, sex, gambling or television. Internet addiction consists of gaming, shopping, and gambling to social networking (Block, 2008; Kuss & Griffiths, 2012). Over the past decades, Internet and game industries have been growing and increasing numbers of people are considered as addicted to Internet and video games. Although Internet gaming disorder has not already seen as a pathologic disorder, it appeared in the appendix of the updated version of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5) for the first time to encourage research about this topic (Kuss, 2013). In the context of new DSM-5,

Internet gaming disorder is associated with the insistent and frequent use of the games on Internet with other players which, then, leads to clinically important damage or distress. Passing excessive time with gaming; withdrawal symptoms such as irritability, anxiety and sadness; increased tolerance; lack of control; use despite negative consequences; mood changes and relapse can be accounted for diagnostic criteria. In addition, clinical studies revealed these overlapping symptoms in Internet addicts. (Kuss, 2013; Lemos et al., 2014; Mehroof & Griffiths, 2010).

Many studies have shown obsessive-like behaviors such as gambling addiction and substance abuse cause changes in brain activity (Irak, Soylu & Çapan, 2016). Therefore, video game addiction may create some significant changes in some cognitive processes, cause long-term behavioral problems, and harmfully influence the natural development of brain (Basak et al. 2008). Although some researches (such as Boot et al. 2008; Coltazo et al. 2013; Ferguson, 2007; Green & Bavelier, 2003, 2015) mention about the positive effects of playing video games on visual spatial perception, attention, and memory, other studies (such as Irak, Soylu & Çapan, 2016; Irons et al., 2011; Powers et al., 2013;) indicate playing video games has no or limited effect on cognitive factors.

According to Gentile et al. (2016) violent video games such as Unreal Tournament or Medal of Honor have high speed, high perceptual and motor load, unpredictability, and emphasis on peripheral processing as well as having violent content. So, training studies using these games demonstrated an effect on perceptual processing such as multiple object tracking, spatial attention, spatial resolution, central and peripheral attention skills (Gentile et al., 2016; McDermott et al., 2014; Sham et al., 2015).

Games could be separated as violent and nonviolent based on contents of games. As is known, today's most popular video games have violent content (Irak, Soylu & Çapan, 2016). Violent video game addiction can result in some threats such as problems in psychological well-being (Gentile et al., 2016; Weber et al., 2006) as well as it creates some significant changes on cognitive processes (Bartholow, Sestir & Davis, 2005) and brain functions of the individuals (Mathews et al., 2005; Wang et al., 2009). However, findings of the studies are inconsistent in terms of negative and positive effects of violent game addiction on individuals' brain development, cognitive and psychological conditions. From this point of view, importance of examining the possible changes after long-term exposure to violent games in cognitive, behavioral and neurobiological functions increases. Weber et al. (2006) questioned whether violent has an impact on aggression (which is named "effect hypothesis") or people with aggressive tendencies prefer to play violent video games (which is named "selection hypothesis"). Many studies (such as Bartholow et al., 2006; Griffiths, 2000; Mathews et al., 2005) demonstrated a correlation between violent game playing and aggressiveness. Mathiak et al. (2011) stated that although some violent video game players might be desensitized to real world violence, lose their empathy and become more aggressive, there are some other studies (such as Elson & Christopher, 2013) showed no much negative effect, especially in terms of aggressiveness. Wells (2002) indicated that individuals may demonstrate memory bias if different emotional stimuli are corresponding their emotional states which means that emotional content could have an effect on individuals' cognitive performances both negatively or positively. Therefore, excessive and persistent exposure to emotionally violent stimuli, as in violent game addiction, might cause to modifications in cognitive processes even if the individuals do not have any

cognitive problems. Therefore, the goal of this study is to investigate the effects of playing violent video games on working memory.

1. 1. 1. Neurobiology and Neuroimaging of Game Addiction. Growing number of neuroimaging techniques have been employed in the Internet game addiction studies. These studies help to analyze the addiction correlates in terms of brain functions and structures (Kuss, 2013). Neuroimaging studies proposed that Internet and game addiction have common features in brain as such in substances related addiction and pathological gambling (Kuss & Griffiths, 2012). According to the results, individuals with symptoms of Internet or game addiction exhibit greater activation in some brain regions which are related to reward, addiction, craving, and emotion during gaming and even presentation of the game cues (Han et al., 2011; Mathiak et al., 2011; Kuss, 2013). In accordance with that, increased activations were observed in some brain regions such as the nucleus accumbens (mostly right dominant), amygdala, anterior cingulate, basal ganglia, dorsolateral prefrontal cortex, right caudate nucleus, right orbitofrontal cortex, insula, premotor cortex, precuneus, and thalamus (Han et al., 2011; Irak, Soylu & Çapan, 2016; Takeuch et al., 2016).

EEG have been used to understand the neuronal correlates of Internet and gaming addiction besides PET scans, fMRI, sMRI, SPECT, and PDR (pupillary dilation response). In the event-related potential (ERP) literature, the amplitude of the P300 component of the ERP, has been often associated with working memory updating (Donchin & Coles, 1988). In several EEG studies (such as Bartholow et al., 2006) examining between violent video game playing and brain function revealed decreased proactive executive control and increased emotional desensitization to violence among chronic violent video game players (Gentile et al., 2016).

Participants attending these studies were exposed to neutral, violent, and nonviolent pictures. Violent video game players had smaller amplitude and increased latency of P300 component when they saw real-life pictures with violent content compared to nonplayers of video game. In another study with similar paradigm, (Engelhardt et al., 2011), participants were divided according to their exposure before the experiment, namely heavy users of video games and low users of video game. Although heavy users did not produce affected P300 amplitude during the exposure to violent pictures, low users showed smaller P300 compared with participants exposed to a nonviolent video game. The authors suggested that emotional content of the stimulus could produce desensitization even in a single exposure to violent video game (Arriaga et al., 2015)

Mathews et al. (2005)'s study demonstrated that adolescents exposed to high media violence showed reduced frontal lobe function during the performance of executive function. Since violence had been related with aggressiveness, they expected to see reduced frontal lobe activation in subjects who exposed to high media violence as similar to aggressive individuals. Consistently, their results demonstrated that all subjects with exposure to high media violence exposure showed activation as subjects with disruptive behavioral deficit did. Thus, they suggested that high exposure to media violence could have an effect on brain functioning even if in the absence of aggression trait. According to Bartholow et al. (2006), this finding was in accordance with the other studies exhibiting executive dysfunction among violent video game players, since working memory updating was considered as a key element of executive cognitive function. In their study, violent video game players showed reduced P300 amplitude and increased P300 latency compared to nonviolent video game players. Thus, results suggested that P300 was

an indicator for level of evaluative categorization during processing of emotionally relevant stimuli (Bartholow et al., 2006). Moreover, Yu et al. (2009) proposed that excessive Internet use led to significant smaller amplitude and longer latency values of P300 in all electrode regions which suggested that excessive Internet use had a negative influence on information coding and integration in the brain.

1. 2. Video Games and Working Memory

Baddeley and Hitch (1974) suggested that working memory refers to a kind of system which is responsible for both sensory information storage and modality-specific processing in complex cognitive processes. This "work space" functions as a temporary storage and includes three components for maintenance of verbal information, called the phonological loop, visuospatial information called the visuospatial sketchpad, and a higher-order functioning, namely the central executive (Kensinger & Corkin, 2003; Schröger, Mecklinger & Pollman, 2004; Hambrick et al., 2010). Years later, a fourth component was added to the working model called the episodic buffer. It was a kind of system that gets information from the subsidiary systems and long-term memory, and takes them in a temporary storage as united episodic representations (Baddeley, 2000).

The working memory is mostly located in the dorsolateral prefrontal lobe of the brain. Working memory inevitably becomes functional during recall and recognition via long-term memory. Posterior regions are the storage buffer of working memory for the spatial location and visual objects. Verbal information has been lateralized to the left while spatial information to the right. Anterior cortex, especially ventral part of prefrontal cortex is related with storage/retention of

information in the working memory, and is related to ventral of the anterior cortices, especially PFC. It is also lateralized according to the content (Yıldırım, 2003).

As aforementioned, working memory is related with sensory information storage. Thus, it can be said that emotional stimuli could affect its performance. In a study using a modified match-to-sample task conducted by Perlstein, Elbert and Stenger (2002), it was revealed that emotional content of stimuli modifies working memory processes. According to the results, working memory related brain activation decreased following a negative emotional content which suggested that emotional content might hamper the performance on working memory tasks (Kensinger & Corkin, 2003). According to this, since people with violent video game addiction were exposed to negative emotional content for a great amount time, they could be expected to show decreased working memory performances.

Previous studies investigated the relationship between video game playing and working memory functions but they showed inconsistent results. During video game playing, participants need to keep their goal in mind, remember the information given and perform their task. Especially in strategy and first-person shooter games, gamers should remember the locations in a large map, complete their mission while distinguishing their enemies from other team members (Boot et al., 2008). Mahncke et al. (2006) claimed that playing video games may improve working memory ability due to the fact that players need to store and remember many stimuli at the same time in order to be successful during playing games. In a study conducted by Boot et al. (2010), participants playing video games less than 3 h a week, were trained for a new video game and they demonstrated greater working memory performances after the training. Cross-sectional study conducted by Sungur and Boduroğlu (2012) also proposed that action video game players outperformed on

tasks of working memory. Coltazo et al. (2013) had similar findings in which they observed increased working memory performance of game players relative to non-players.

Shams et al. (2015) conducted a meta-analysis to investigate the effects of video game playing on cognition and brain structures. They examined many studies which were conducted with gamers, non-gamers and expert gamers who had played different game-genres. One of the studies implemented a visual working memory task and they found significantly larger gray matter volume of frequent and expert game players in regions associated with memory like right posterior parietal cortex. Nouchi et al. (2013)'s study using a training game (Brain Age) and a puzzle game (Tetris) revealed that participants in the training group showed improvements in executive functioning, working memory, and processing speed (as cited in Shams et al., 2015). The results of these studies were consistent with other studies which claimed affected working memory performance. In another study using five game genres (action game, spatial memory game, match-3 game, hidden-object game, and an agent-based life simulation game (control)), they found that different game genres cause improvements in different cognitive processing, such as spatial working memory.

On the other hand, Powers et al. (2013) conducted a meta-analysis to investigate the effects of video game play on cognitive processes and demonstrated no evidence about positive influence of playing video games on working memory. In addition, Boot et al. (2008) found no difference in terms of working memory performances between expert video game players and non-players.

1. 3. The goal of the present study

Previous studies have investigated that violent video game play led to improvements in working memory (Blanker et al., 2014; Boot et al., 2008; Colzato et al., 2013; Mahncke et al., 2006; Sungur & Boduroğlu, 2008), but the contradictory results do not provide a clear answer (Irak, Soylu & Çapan, 2016; Irons et al., 2011; Powers et al., 2013; Wilms et al., 2013).

Blacker et al (2014) demonstrated improvement in capacity of visual working memory using a training method. Participants trained with action video game playing showed better performances than other participants trained with control games in a change detection task which was employed to measure the visual working memory. Accordingly, Colzato et al. (2013) demonstrated that experienced violent video game players had better working memory performances compared to control group.

On the other hand, Wilms, Petersen and Vangkilde (2013) revealed that excessive action video game playing led to enhancement in the encoding speed to short-term memory, whereas there was no change in visual short-term memory capacity and visual attention threshold. They interpreted these results as such that experienced gamers had improved skills on using the limited capacity of short-term memory more quickly and proficiently. Moreover, Irak, Soylu and Çapan (2016) found decreased working memory performances in violent video game addicted compared to control group.

These contradictory results warrant additional research into the causal link between playing video game play and working memory processes. Furthermore, there is an extensive body of literature documenting a relationship between video game addiction and several cognitive processes which we briefly mentioned above. Notably, understanding the effects of violent video game addiction on working

memory attention may provide a guide for researchers, educators and parents about cautions or enhancements.

Working memory seems to have an important role in the video game because participants need to keep their goal, remember the information given and perform thetask. Especially in strategy and first-person shooter games, gamers should remember the locations in a large map, complete their mission while distinguishing their enemies from other team members (Boot et al., 2008). Relatedly, visual working memory is essential in order to gain new abilities and knowledge and solve unusual tasks. Since visual working memory helps sustaining attended information across saccades and other visual interruptions, relating objects or scenes regarding visual characteristics, and navigating the visual world, more researches are needed to find useful method to expand this process (Blanker et al, 2014). Therefore, it was expected that violent video game addicted showed improved working memory performances than non-player control group.

Likewise, since existing studies were conducted by using mostly fMRI technique, studies on event-related potentials event-related potentials are not sufficient (Irak, Soylu & Çapan, 2016). In the event-related potential (ERP) literature, since ERP studies were generally focused on a variety of cognitive performances at the same studies, studies on specific cognitive processes were needed. Moreover, there are limited studies on working memory performance in violent game addiction. EEG study give a chance to take a deeper look at differences in brain responses and to make comparisons between game-addicted and non-player groups in terms of working memory performances.

Additionally, there are some methodological issues to be concern which are lack of accepted inclusion and exclusion criteria for addicted group. To eliminate this

problem, three psychological measurements were used to clearly divide the addicted and non-player group.

In sum, the current study sought to address the following questions: (a) Are there any differences between ERP responses of violent video game addicted and non-players under working memory task? (b) Is there a relationship between amplitude and latency values of ERPs under working memory task, and behavioral variables (accuracy of responses, confidence level and reaction time)? (c) If there is, how similar or different are these relationships between violent video game addicted and non-players and (d) Does violent video game addiction enhance or decrease the working memory performances?

Thus, following hypotheses will be tested:

1. Violent video game addicts show better performances on working memory task than non- players.
2. Violent video game addicts are faster in responses than non-players.
3. Amplitude and latency values of ERPs are different in violent video game addicts compared to non-players according to resolvable and misleading trial types of probe-1 and probe-2 phases.
4. ERP responses of violent video game addicts under working memory task are different compare to non-players.

CHAPTER 2

METHOD

2. 1. Participants

40 students attending Bahçeşehir University recruited for the study. The data from the remaining 40 participants (16 female) aged between 18 and 31 (*M* = 22.40 *SD* = 3.00) was included. Participants who did not have normal or corrected-to-normal vision, and with usage of any kind of psychiatric and/or neurological medication, with history of neurological, psychological, or memory diseases were excluded from the study.

Participants were included based on the time they spend for violent game playing per week, DSM- based pathological game addiction symptoms (Gonnerman & Lutz, 2011), and their scores on the Game Addiction Scale developed by Lemmens et al. (2009). The first criterion was playing violent video games more than 16 h/week, the second criterion was reporting more than three symptoms on the Pathological Game Addiction Symptoms List and the last criterion was getting more than 55 total score on the Game Addiction Scale for the namely addicted group. For the control group, the first criterion was not having any experience with any type of video games, the second criterion was obtaining a less than 37 total score on the

Game Addiction Scale, and the last criterion was reporting no symptoms on the Game Addiction Symptoms List for the control group. According to the results, participants were separated into two groups which are namely addicted (video game addicts) and control group (non-players).

2. 2. Materials

Each participant received an informed consent form, a demographic questionnaire and research materials. Research Materials included Pathological Game Addiction Symptoms List (Gonnerman & Lutz 2011), Game Addiction Scale (Lemmens et al., 2009) and Working Memory Task (Harkin & Kessler, 2009).

2. 2. 1. Pathological Game Addiction Symptoms List. The original list was created from the pathological gambling symptoms according to DSM-IV criteria by Gonnerman and Lutz (2011). In Pathological Game Addiction Symptom List, the word "gambling" was transformed to the word "gaming" in the sentences for the present study. The list also transformed as "yes-no" questions type. Turkish adaptation of the list was developed by Arslan-Durna (2015) and Başer (2015) and it had 16 items. The scores obtaining from the list ranged from 0 to 16 (Irak, Soylu & Çapan, 2016).

2. 2. 2. Game Addiction Scale. Game Addiction Scale was originally settled by Lemmens et al. (2009) to measure the degree of game addiction. It was 21-item scale with seven factors. The factors were salience, tolerance, mood modification, relapse, withdrawal, conflict, and problems. Participants' responses could be given on a 5-point Likert scale from 1 (never) to 5 (very often). Turkish adaptation was developed by Arslan-Durna (2015) and Başer (2015) (Irak, Soylu & Çapan, 2016).

Participants take minimum 21, and maximum 105 points from the scale. High scores show high levels of game addiction.

2. 2. 3. Working Memory Task. The task was adapted from Harkin and Kessler (2009). There are four consecutive phases (see Figure 1). Four capital letters were used as stimuli and they were shown against a gray back-ground within a 2 (columns) by 3 (rows) matrix covering an area of 300 × 420 pixels. In the first (namely; encoding) phase, participants were shown a 1000-ms fixation cross and then, four letters were presented randomly in four of the six possible locations. Participants had 1000 ms for encoding the identity and the location of each letter. After 500 ms, in the second phase (namely; probe-1) participants were instructed to show the location of a given letter via mouse and to have only 2000 ms to answer. In this phase, two types of trials were created according to whether the probe-1 letter had (namely; resolvable) or had not been (namely; misleading) part of the encoded set. In experimental group, probe-1 was omitted in order to measure working memory performance on the primary task under ideal conditions. Probe -1 and probe-2 were parted by a 500-ms interval. Intermediate probe-1 was not included in the experimental group. Thus, participants were exposed to a gray screen during 5500 ms between encoding and probe-2. The third phase (namely, probe-2) was the actual memory test for each trial. In this phase, participants were instructed to show whether a letter would be correctly located regarding the originally encoded set (2000 ms). In all of these trials, the probe-2 letter's identity belonged to encoded set. On the other hand, the probe location was correct only on 50% of the trials. Finally, in the fourth phase, participants were shown a scale and instructed to indicate their

degree of confidence in their probe 2 response (6 levels: 1 = totally certain to 6 = totally uncertain).

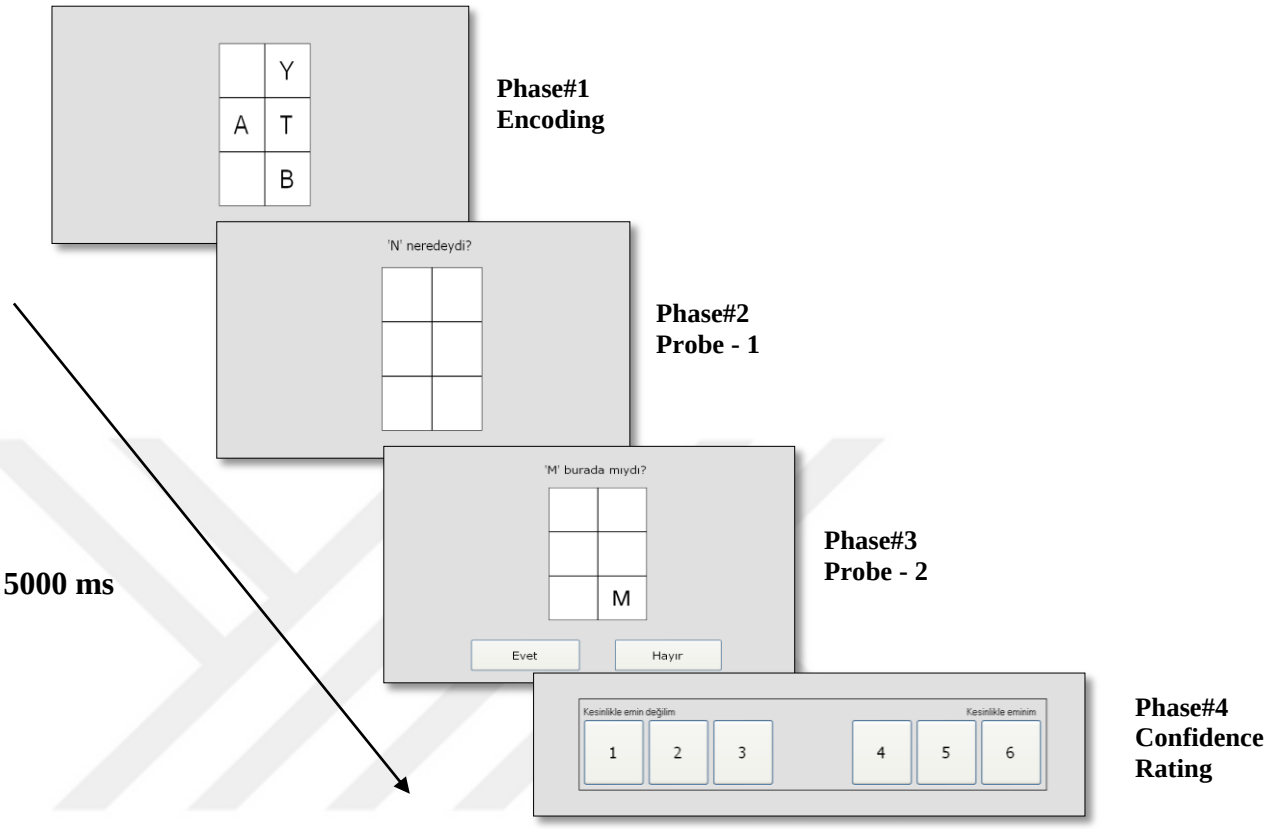

Figure 1. Phases of Working Memory Task.

2. 3. Electrophysiological Recording and Processing.

Working memory task was conducted on an electrically shielded and soundproof room, and ERPs were recorded during the working memory task. 32 channels EEG/EP NeuroScan system was used to present stimuli, and to record, store and analyze the electrophysiological data. EEG activity was recorded with 30 electrodes inserted in elastic Quick-caps (Neuromedical Supplies, Compumedics, Inc., Charlotte) in line with the international 10-20 system.

EOG activity was also measured from two bipolar channels which were placed at the outer canthus of each eye, and below and above the left eye. Moreover, additional electrodes were fitted in BP1/BP2, and placed on the left and right

mastoids (M1/M2). All EEG electrodes were referenced on-line to an electrode at vertex and re-referenced off-line to linked mastoids. EEG/EOG signals were amplified and recorded at 1000 Hz sampling rate by using Synamp2 amplifier at AC mode (Neuroscan, Compumedics, Inc., Charlotte) with high-pass and low-ass respectively: filter set at 0.15 and 100 Hz. EEG electrode impedance was kept below 5 kΩ.

Edited 4.5 (Neuroscan, Compumedics, Inc. Charlotte) was applied to all participants' data set to EEG data pre-processing. Data was down-sampled to 250 Hz in order to decrease computational loads. Then, data were low-pass filtered at 30 Hz and high-pass filtered at 0.15 Hz. The segmentation was extracted with an interval of -200 ms preceding and 1000 ms following the prime face onset. Artifact refection was planned to perform in two steps. Firstly, vertical and horizontal EEG/EOG channels encompassing activity which exceeded a threshold of ±100 μV were automatically detected and rejected. Secondly, trials with saccades identified over the horizontal EOG channel were manually removed. By reason of the calculation of ERPs, artifact-free segments were baseline corrected using 100 ms pre-stimulus period and then averaged for working memory task.

2. 4. Electrophysiological Analyses

Event Related Potentials (ERPs) were analyzed via mean analysis (averaging) in the current study. The average of ERPs was determined in temporal direction. Grand averages were calculated separately for frontal electrodes namely, F3, F4, Fz, FC3, FC4 and FCz. Latency (ms) and amplitude (μV) values of the peaks were detected for each participant and each electrode. Naming of these peaks were based on their order (1 to 4) and their polarity (P: positive, N: negative): N1, P1, P2, N4, P3, late negative potentials (LNP) and late positive potentials (LPP).

Peak values were measured separately for resolvable and misleading trials of both probe-1 and probe-2 phases by finding the most positive –or negative point in a given time window. Time windows for components were respectively defined as P100 for the most positive point between 50 and 100 msec, N100 for the most negative point between 80 and 150 msec, P200 for the most positive point between 130 and 200, P300 for the most positive point between 200 and 300, LNP for the most negative point between 600 and 750 msec, and LPP for the most positive point between 600 and 850 msec. These time ranges sufficiently included the latency variations of the ERP peaks that were obtained under all conditions of the experimental paradigms. Although statistical analysis was carried on previously mentioned electrode side, topographies were investigated for 30 electrodes: C3, C4, Cz, CP3, CP4, CPz, F3, F4, F7, F8, Fz, FP1, FP2, FC3, FC4, FCZ, FT7, FT8, T7, T8, TP7, TP8, O1, O2, Oz, P3, P4, P7, P8 and Pz, representing five brain regions: central, frontal, occipital, temporal and parietal. ERP topographies for the 30 electrodes are presented for each experimental condition.

2. 5. Procedure

The study was approved by the Bahçeşehir University Ethics Committee, before the experiment. Participants were selected via a survey which includes questions about time they spent for violent game playing per week and which games they played most. According to the survey results, participants fitting the inclusion criteria were contacted and informed about the study. Participants had interviews with the researcher. During interviews, participants were asked survey questionnaire again to be sure they fit into the study. Then, 40 participants were given appointments for the experiment. Moreover, they were warned to sleep well in the night, to have breakfast, not to drink coffee or energy drinks before coming to the

experiment in order to prevent possible confounding. Participants were informed about the aim, benefit and potential risks of the study.

The study was conducted in the Brain and Cognition Research Laboratory of Bahçeşehir University. The Brain and Cognition Research Laboratory is equipped with state-of-the art facilities to carry out research in the neurobiological mechanisms of human cognitive processing. Working Memory Task was given in a single session and same hours every day using an IBM compatible 15-inch computer running Windows XP. All applications including electrode cap preparation, measurements and the experiment took almost 1-1.5 hours for each participant. Participants were provided a hair care service located in the campus to be cleaned from the gel that was used in the experiment. This was the standard procedure for the whole electrophysiological experiments in the research laboratory. Participants were given 20 TL gift-card, after they completed the study.

CHAPTER 3

RESULTS

Data were screened to understand whether there was any missing value and outlier. Then the assumptions of multivariate statistics such as normality, linearity and homescedasticity were examined before the statistical analysis. There were no univariate or multivariate outliers (Tabachnick & Fidell, 2007). The results of the study were reported in this order: behavioral results, visual inspection results, and lastly ERPs results.

3. 1. Behavioral Results

Independent sample t - tests were conducted to compare correct and incorrect responses; and reaction times of addicted and control groups in probe - 1 and probe - 2 trials. There was no significant difference in correct and incorrect responses of addicted and control groups in probe - 1 and probe -2 trials.

There was no significant difference in reaction times for correct and incorrect responses in addicted and control groups in probe-1 trial.

For the reaction time for correct responses of addicted and control groups in probe - 2 trial, homogeneity of variances was not equal, $p < .05$. On the other hand, there was a significant difference in reaction time for incorrect responses in addicted (M = 1527, SD = 633, 52) and control (M = 2462, SD = 864, 71) groups in probe - 2 trial; t (15) = 3, p = .021. (see Table 1)

Table 1.

t - test Results Comparing Addicted and Control Groups on Working Memory Performance.

		Addicted		**Control**		
		M	*SD*	M	*SD*	*t*-test
Probe-1	Correct Responses	30	.94	29,4	.99	ns
	Incorrect Responses	1,8	2	2	1,07	ns
Probe-2	Correct Responses	28,9	1,66	29,1	1,28	ns
	Incorrect Responses	1,3	.68	1,9	1,6	ns
Probe-1	Correct Responses RT	1607	259	1691,4	287	ns
	Incorrect Responses RT	2844,44	1178,61	2732,53	1157,4	ns
Probe-2	Correct Responses RT	1721,22	390,13	1781,47	189	ns
	Incorrect Responses RT	1527	633,52	2462	864,71	3.00*

*$p < .05$

There was no significant difference in confidence level for correct and incorrect response; and reaction times of addicted and control groups. (see Table 2)

Table 2.

t - test Results of Confidence Levels Comparing Addicted and Control Groups on Working Memory Performance.

	Addicted		**Control**		
	M	*SD*	M	*SD*	*t*-test
Correct Response	5,96	.06	5,93	.10	ns
Incorrect Response	5,3	1,06	5,38	1,16	ns
Reaction Time	745,6	226	836,1	175,53	ns

*$p < .05$

Results showed that although homogeneity of variance was satisfactory for all variables except incorrect responses and reaction time for correct responses in probe - 2, groups did not significantly differentiate on any working memory scores, $p > .05$. However, groups significantly differentiate on working memory scores in terms of reaction times for incorrect response in probe - 2 trial.

3. 2. ERP Results

3. 2. 1. Visual Inspection Results. Figure 2 and 3 show the grand average filtered waveforms during working memory task phases in terms of its resolvable and misleading trials. The waveforms were visually examined to understand peaks of interest and their amplitude and latency values. The latency and amplitude of each ERP component were analyzed at the electrode sites where they reach the maximum amplitudes, but not at electrodes with suboptimal amplitudes or in which a certain component is not clearly seen. Thus, even though topographies of all 30 electrodes were given in figure 2 and 3, since working memory process is heavily related with

frontal lobe function (Courtney et. al, 1998; Nissim et. al, 2017), the main focus of the statistical analyses was on 6 frontal electrode sites namely, F3, F4, Fz, FC3, FC4 and FCz. Latency (ms) and amplitude (μV) values of the peaks were detected for each participant and each above mentioned six frontal zone electrodes. Peak values were measured separately for resolvable and misleading trials of both probe-1 and probe-2 phases by finding the most positive –or negative point in a given time window.

In terms of probe-1 phase, negative components of N100, N200 and N400 followed by positive components of P100, P200, P300 and LPP were observed at almost all electrode sites but in different amplitude and latencies. It was recorded that there was a slighter trend of N100 peak at frontal-left and frontal-central electrode sides than frontal-right electrode sides. In terms of both resolvable and misleading trials, it was observed that addicted group' amplitude of N100 peak was higher than control group at frontal electrode sites, whereas it was vice versa at occipital electrode sites. The latency value of N100 was earlier for control group. It was observed that addicted group produced higher amplitude value of P100 peak than controls in both resolvable and misleading conditions. However, the latency values were earlier in controls than addicted group. Additionally, it was detected that control group produced higher P200 components at frontal central electrode sides than addicted group in terms of misleading trial, although addicted group produced higher amplitudes than control group in both conditions at occipital and parietal electrode sites. P300 components at posterior frontal electrode sides were higher in amplitude than at anterior frontal sides. Group differences were more observable at right frontal electrode sides. It was recorded that addicted group produced higher amplitude value of P300 at frontal and parietal electrode sites. The pattern was

clearer for addicted group at occipital electrode sites. According to this, it could be said that amplitude values of addicted group in resolvable trial was higher than misleading trial in terms of P300 components.

In terms of probe-2 phase, negative components of N100 followed by positive components of P100, P200, P300 and LPP were observed. In general, it was observed that addicted group produced higher amplitude values of N100 at almost all frontal electrode sites than control group in resolvable trials. Moreover, addicted group produced higher amplitude values in misleading trials at anterior frontal electrode sites. The latency of N100 was earlier for control group than addicted group in resolvable trials. However, the amplitude values of control group were higher than addicted group in both conditions at parietal and occipital electrode sites. The amplitude value of P100 peaks were higher for control group for both conditions at left and centro-parietal electrode sites, while it was higher for addicted at right parietal electrode sites. Amplitude values of P200 were higher for addicted than controls in both conditions at occipital and parietal electrode sites. However, it was recorded that control group had higher amplitude value of P200 components than addicted group at frontal electrode sites. In misleading trials, group differences were more observable in terms of P200 components at frontal electrode sites. The latency differences of P200 components between groups were clearly observable in misleading trials. According to this, the latency of P200 was earlier for control group than addicted group.

Moreover, it was recorded that addicted group produced greater P300 amplitude than control group in resolvable trials at frontal, central-occipital and parietal electrode sites. The latency was earlier for addicted group at occipital electrode sites which was also corresponding with statistical analyses. In misleading

trials, control group produced higher P300 amplitude than addicted group at frontal, occipital and parietal electrode sites. Finally, it was observed that control group produced higher amplitude and earlier latency of LPP (especially P600) than addicted group in misleading trials.

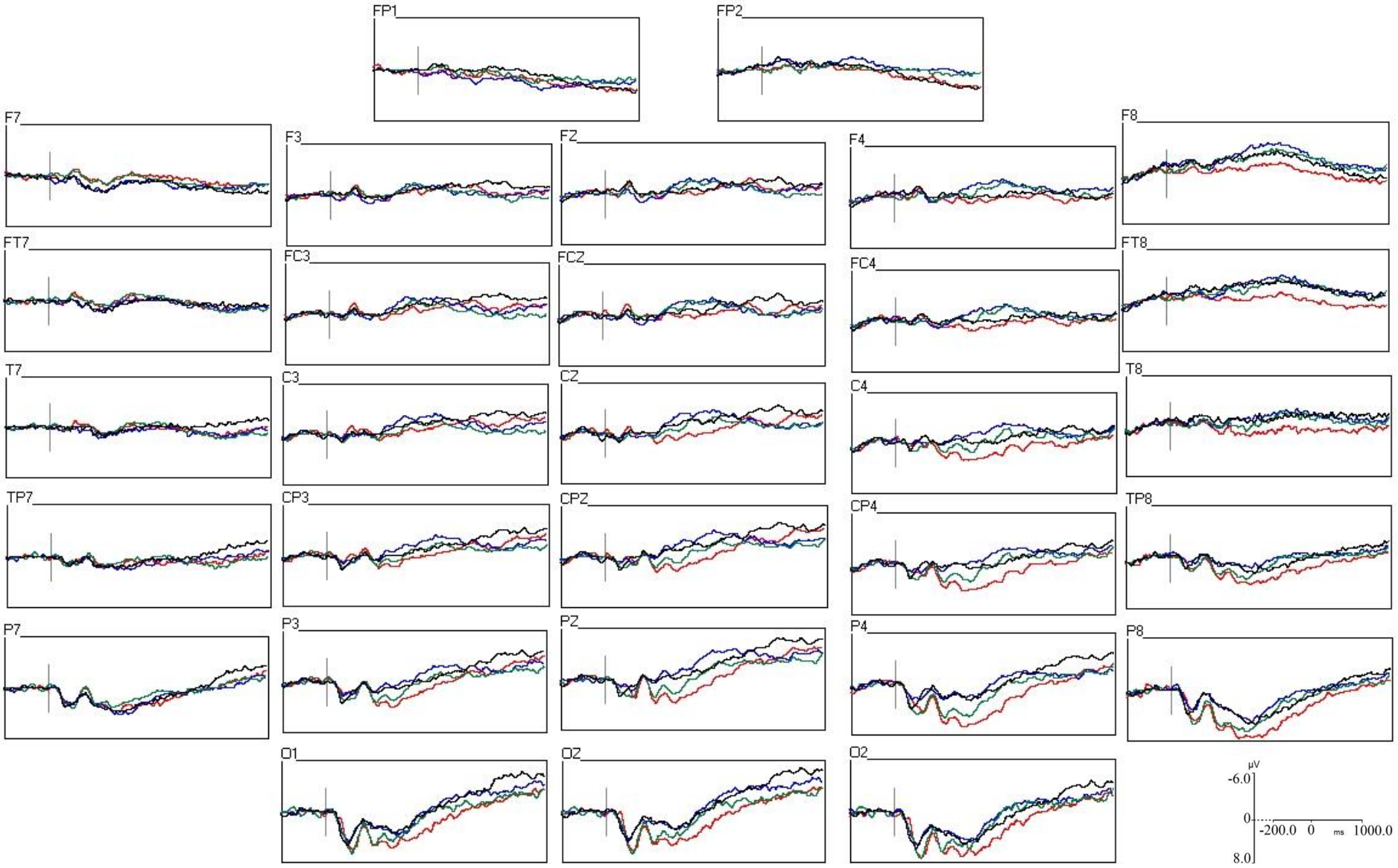

Figure 2. ERP grand average waveforms of 30 electrodes during Probe-1 Resolvable vs. Misleading Trials of Addicted and Control Groups

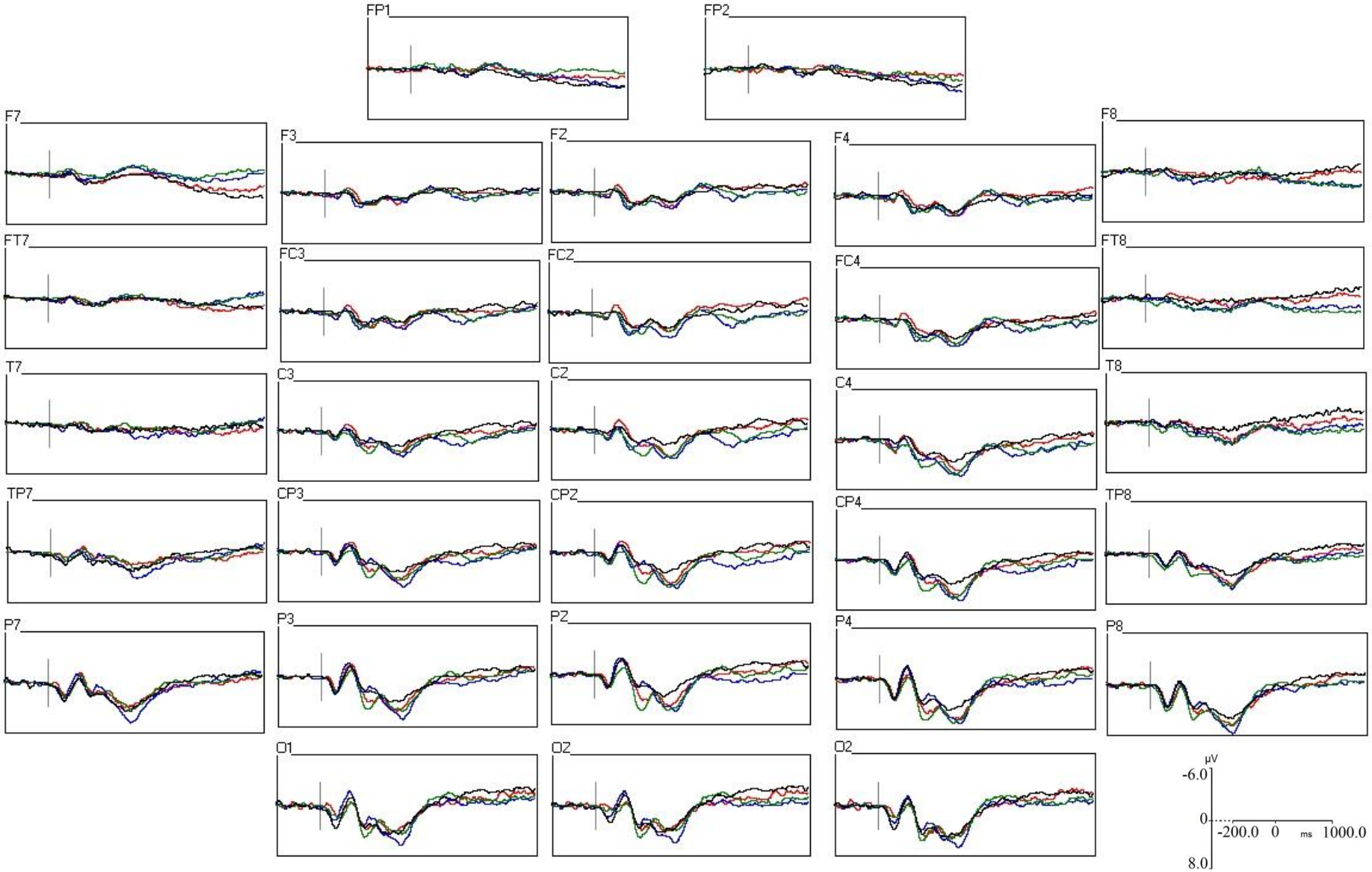

Figure 3. ERP grand average waveforms of 30 electrodes during Probe-2 Resolvable vs. Misleading Trials of Addicted and Control Groups

3. 2. 2. Statistical Analysis. Analyses were conducted for resolvable and misleading conditions of probe-1 and probe-1 phase separately. Amplitude and latency values of N100, N400, P100, P200, P300, LNP and LPP peaks were compared between control and addicted groups via repeated measures ANOVA. Statistical analysis was conducted for two different electrode cluster separately (Frontal: F3, F4 and Fz; Posterior Frontal: FC3, FC4 and FCz). Therefore, electrode location and groups were independent variables, while amplitude and latency values of ERP peaks were dependent variables for repeated measure ANOVA. The factors for EEG analyses were electrode locations and trial type and groups. According to this, 2 separate- 2 (trial type: resolvable and misleading) x 3 (location: F3/FC3, F4/FC4, Fz/FCz) repeated measures ANOVAs were performed. Greenhouse Geisser correction was used in the condition of violated sphericity assumption. To inflate Type 1 error Bonferroni confidence interval adjustment was employed and α value was 0.05 for each p. (Tabachnick & Fidell, 2007). Detailed values of mean and standard errors and significant ANOVA results for amplitude and latency values are given in Appendix A and B.

3. 2. 2. 1. Amplitude comparisons of ERPs in Probe-1 Resolvable vs. Misleading Trials. Figure 4 and 5 show the waveforms of 6 electrodes during probe-1 and probe-2 phases for resolvable and misleading trials.

The results indicated a significant trial type effect on N100 amplitude (Wilks' Lambda = .935 F (1, 38) = 7.002, $p < .05$, $\eta^2 = .16$) and N400 amplitude (Wilks' Lambda = .764 F (1, 38) = 11.273, $p < .05$, $\eta^2 = .24$) at anterior frontal electrodes. Post hoc analysis indicated that N100 amplitude in sesolvable trial (M = -2.95, SE = .35) was significantly higher than misleading trial (M = -1.97, SE = .36). N400

amplitude in misleading trial (*M* = -4.37, *SE* = .5) was significantly higher than resolvable trial (*M* = -3.19, *SE* = .52). For posterior frontal electrodes, N100 amplitude in resolvable trial (*M* = -2.78, *SE* = .41) was significantly higher than misleading trial (*M* = -1.76, *SE* = .4). N400 amplitude in misleading trial (*M* = -4.84, *SE* = .59) was significantly higher than resolvable trial (*M* = -3.43, *SE* = .55).

The interaction effect between location and trial type on amplitude values was significant for N400 amplitude (Wilks' Lambda = .788 *F* (2, 38) = 4.964, $p < .05$, $\eta^2 = .21$), LNP (Wilks' Lambda = .525 *F* (2,37) = 16.766, $p < .05$, $\eta^2 = .48$) and LPP (Wilks' Lambda = .538 *F* (2,37) = 15.892, $p < .05$, $\eta^2 = .46$) at anterior frontal electrodes. Paired-sample *t*-test was conducted in order to analyze difference between electrode sites. According to results, N400 amplitude in misleading trial at F4 electrode site (*M* = -3.94, *SE* = .43) was significantly higher than resolvable trial at F4 electrode site (*M* = -3.51, *SE* = .6) and in misleading trial at Fz electrode site (*M* = -4.38, *SE* = .58) was significantly higher than resolvable trial at Fz electrode site (*M* = -3.45, *SE* = .54). LNP amplitude in misleading trial at F4 electrode site (*M* = -3.57, *SE* = .59) was significantly higher than resolvable trial at F4 (*M* = -2.5, *SE* = .62). LPP amplitude in misleading trial at F3 electrode site (*M* = 2.39, *SE* = .5) was significantly higher than resolvable trial at F3 electrode site (*M* = 1.27, *SE* = .59).

The results indicated a significant trial type effect on N100 amplitude (Wilks' Lambda = .858 *F* (1, 35) = 5.775, $p < .05$, $\eta^2 = .14$) and N400 amplitude (Wilks' Lambda = .694 *F* (1, 35) = 15.425, $p < .05$, $\eta^2 = .31$) at posterior frontal electrodes. Post hoc analysis indicated that N100 amplitude in resolvable trial (*M* = -2.78, *SE* = .41) was significantly higher than misleading trial (*M* = -1.76, *SE* = .4). N400 amplitude in misleading trial (*M* = -4.84, *SE* = .59) was significantly higher than resolvable trial (*M* = -3.43, *SE* = .55).

The interaction effect between location and trial type on amplitude values was significant for N400 amplitude (Wilks' Lambda = .776 F (2, 34) = 4.917, p < .05, η^2 = .22), LNP (Wilks' Lambda = .474 F (2,34) = 18.891, p < .05, η^2 = .53) and LPP (Wilks' Lambda = .549 F (2,34) = 13.975, p < .05, η^2 = .45) at posterior frontal electrodes. Paired-sample *t*-test was conducted in order to analyze difference between electrode sites. According to results, N400 amplitude in misleading trial at FC4 electrode site (M = -4.77, SE = .59) was significantly higher than resolvable trial at FC4 electrode site (M = -2.56, SE = .53), and in misleading trial at FCz electrode site (M = -5.13, SE = .62) was significantly higher than resolvable trial at FCz electrode site (M = -3.78, SE = .63). LNP amplitude in resolvable trial at FC3 electrode site (M = -4.2, SE = .7) was significantly higher than misleading trial at F4 (M = -2.85, SE = .65), and in misleading trial at FC4 electrode site (M = -3.69, SE = .51) was significantly higher than resolvable trial at FC4 electrode site (M = -2.74, SE = .58). LPP amplitude in misleading trial at FC3 electrode site (M = 2.32, SE = .58) was significantly higher than resolvable trial at FC3 electrode site (M = .77, SE = .68). The main effect of location on amplitude was not significant.

3. 2. 2. 2. Latency comparisons of ERPs in Probe-1 Resolvable vs. Misleading Trials. According to results, the main effect of location on N400 latency at anterior frontal electrodes (Wilks' Lambda = .750 F (2,37) = 6.177, p < .05, η^2 = .25). Post hoc analysis showed that N400 at F3 (M = 438.1, SD = 10.3) electrode site was significantly earlier in latency than F4 (M = 458.2, SD = 10.2). The main effect of trial type was found significant for LNP latency at anterior frontal electrode sites (Wilks' Lambda = .895 F (1,38) = 4.442, p < .05, η^2 = .11). Post hoc analysis showed that LNP latency in misleading trial (M = 652.15, SD = 9.6) was significantly earlier

in resolvable trial (*M* = 677.96, *SD* = 5.9). Although, the interaction effect between location and group on latency values was significant for N400 amplitude (Wilks' Lambda = .820 *F* (2, 37) = 4.061 $p < .05$, $\eta^2 = .18$), Post hoc analysis showed no significant difference between groups. There was no interaction effect between location and trial type on latency.

The results indicated a significant trial type effect on LNP latency (Wilks' Lambda = .826 *F* (1, 35) = 7.370, $p < .05$, $\eta^2 = .17$) at posterior frontal electrodes. Post hoc analysis showed that LNP latency in misleading trial (*M* = 650.65, *SD* = 8.89) was significantly earlier in resolvable trial (*M* = 67680.58, *SD* = 5.85).

The interaction effect between location, trial type and group on amplitude values was significant for LNP latency (Wilks' Lambda = .748 *F* (2, 34) = 5.735, $p < .05$, $\eta^2 = .25$) at posterior frontal electrodes. However, independent sample *t*-test results showed that there was no significant difference between groups. There was no location main effect or interaction effect between location and trial type on latency. (See Figure 4.)

3. 2. 2. 3. Amplitude comparisons of ERPs in Probe-2 Resolvable vs. Misleading Trials. The results indicated a significant location effect on P300 amplitude (Wilks' Lambda = .816 *F* (2, 37) = 4.172, $p < .05$, $\eta^2 = .18$). Post hoc analysis indicated that P300 amplitude in Fz electrode site (*M* = 4.26, *SE* = .48) was significantly higher than F3 electrode site (*M* = 3.65, *SE* = .40). There was also a significant location effect on LNP amplitude (Wilks' Lambda = .678 *F* (2, 37) = 8.804, $p < .05$, $\eta^2 = .32$). Post hoc analysis indicated that LNP amplitude in Fz electrode site (*M* = -2.3, *SE* = .56) was significantly higher than F4 electrode site (*M* = -1.16, *SE* = .54). Additionally, there was a significant trial type effect on LPP

amplitude (Wilks' Lambda = .873 F (1, 38) = 5.503, $p < .05$, $\eta^2 = .13$). Post hoc analysis indicated that LPP amplitude in misleading trial (M = 3.66, SE = .57) was significantly higher than resolvable trial (M = 2.16, SE = .52).

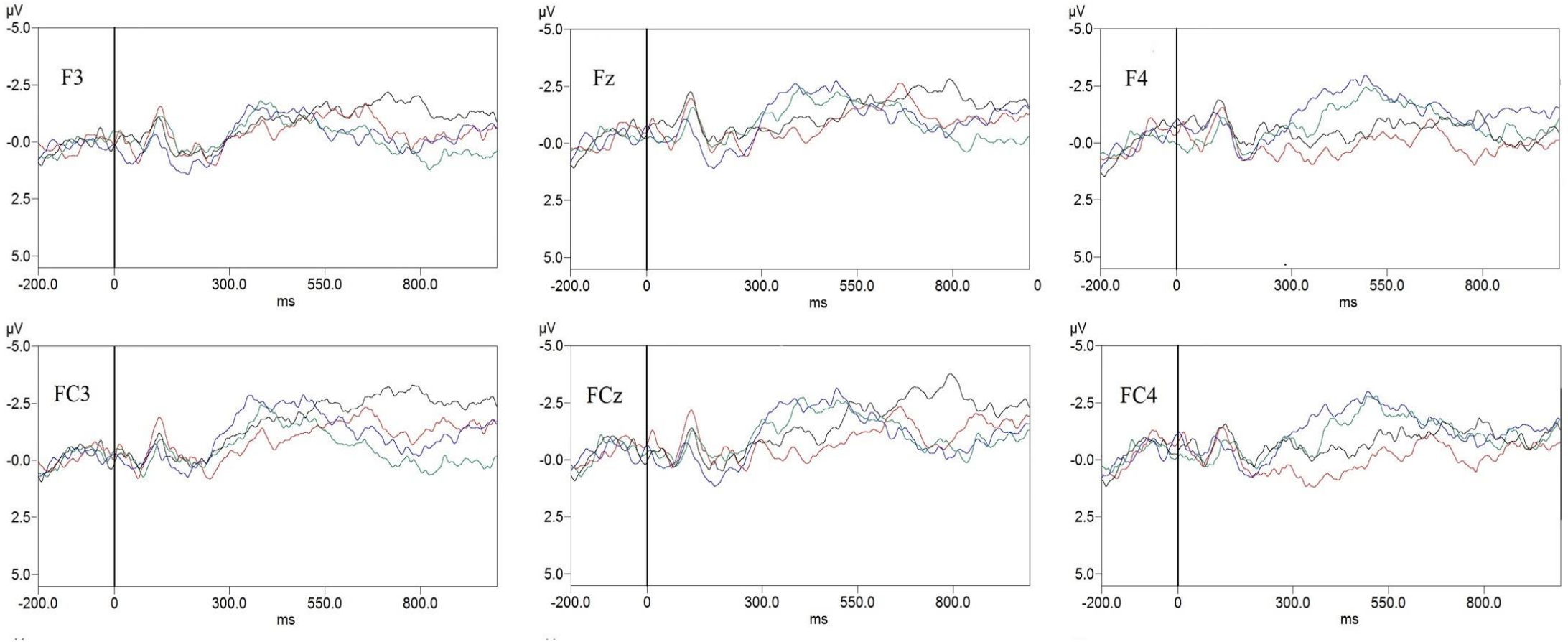

Figure 4. ERP grand average waveforms of 6 electrodes during Probe-1 Resolvable vs. Misleading Trials of Addicted and Control Groups

The interaction effect between location and trial type on amplitude values was significant for P200 amplitude (Wilks' Lambda = .829 F (2, 37) = 3.827, $p < .05$, η^2 = .17) at anterior frontal electrodes. Paired-sample t-test was conducted in order to analyze difference between electrode sites. According to results, P200 amplitude in misleading trial at Fz electrode site (M = 4.69, SE = .48) was significantly higher than resolvable trial at Fz electrode site (M = 3.28, SE = .52).

The results indicated a significant location effect on N100 (Wilks' Lambda = .776 F (2, 37) = 5.344, $p < .05$, η^2 = .22), P200 (Wilks' Lambda = .810 F (2, 37) = 4.341, $p < .05$, η^2 = .19), N400 (Wilks' Lambda = .702 F (2, 37) = 7.841, $p < .05$, η^2 = .30), and LNP amplitude (Wilks' Lambda = .732 F (2, 37) = 6.758, $p < .05$, η^2 = .27) at posterior frontal electrodes. Post hoc analysis indicated that N100 amplitude at FCz electrode site (M = -2.2, SE = .40) was significantly higher than FC4 electrode site (M = -1.31, SE = .38). P200 amplitude at FC4 electrode site (M = 4.68, SE = .44) was significantly higher than FC3 electrode site (M = 4.03, SE = .42). N400 amplitude at FCz electrode site (M = -2.54, SE = .63) was significantly higher than FC4 electrode site (M = -1.2, SE = .51). LNP amplitude at FCz electrode site (M = -2.50, SE = .63) was significantly higher than FC3 (M = -1.33, SE = .52); and LNP amplitude at FCz electrode site was significantly higher than FC4 electrode site (M = -1.33, SE = .51).

The results indicated a significant trial type effect on P200 (Wilks' Lambda = .867 F (1, 38) = 5.806, $p < .05$, η^2 = .13) and LPP amplitude (Wilks' Lambda = .845 F (1, 38) = 6.961, $p < .05$, η^2 = .16) at posterior frontal electrodes. Post hoc analysis indicated that P200 amplitude in misleading trial (M = 5.08, SE = .52) was significantly higher than resolvable trial (M = 3.88, SE = .47). LPP amplitude in misleading trial (M = 4.43, SE = .64) was significantly higher than resolvable trial (M = 2.41, SE = .60).

The interaction effect between location and trial type on amplitude values was significant for N400 amplitude (Wilks' Lambda = .767 F (2, 37) = 5.630, $p < .05$, η^2 = .23), LNP (Wilks' Lambda = .819 F (2, 37) = 4.092, $p < .05$, η^2 = .18) and LPP (Wilks' Lambda = .832 F (2, 37) = 3.738, $p < .05$, η^2 = .17) at posterior frontal electrodes. Paired-sample *t*-test was conducted in order to analyze difference between electrode sites. According to results, LNP amplitude in resolvable trial at FCz electrode site (M = -3.71, SE = .85) was significantly higher than misleading trial at FCz electrode site (M = -1.3, SE = .79). LPP amplitude in misleading trial at FC3 electrode site (M = 4.42, SE = .64) was significantly higher than resolvable trial at FC3 electrode site (M = 2.61, SE = .58); and LPP amplitude in misleading trial at FCz electrode site (M = 4.56, SE = .73) was significantly higher than resolvable trial at FCz electrode site (M =1.62, SE = .81). However, there was no significant difference between groups in N400 amplitude. Finally, there was no interaction effect between location, trial type and group on amplitude.

3. 2. 2. 4. Latency comparisons of ERPs in Probe-2 Resolvable vs. Misleading Trials. According to results, the main effect of location on P300 latency at anterior frontal electrodes (Wilks' Lambda = .801 F (2,37) = 4.603, $p < .05$, η^2 = .20). Post hoc analysis showed that P300 latency at F3 (M = 229.83, SD = 5.7) electrode site was significantly earlier in latency than F4 (M = 240.73, SD = 5.72) electrode site. There was no main trial type or interaction effect between location and trial type on latency.

The results indicated a significant location effect on P100 (Wilks' Lambda = .729 F (2, 37) = 6.862, $p < .05$, η^2 = .27), P300 (Wilks' Lambda = .787 F (2, 37) = 5.020, $p < .05$, η^2 = .21), N400 (Wilks' Lambda = .772 F (2, 37) = 5.437, $p < .05$, η^2 = .23) and LNP (Wilks' Lambda = .851 F (2, 37) = 3.249, $p < .05$, η^2 = .15) latency at

posterior frontal electrodes. Post hoc analysis showed that P100 latency at FC3 (M = 74.49, SD = 3.72) was significantly earlier at FC4 (M = 83.74, SD = 5.18) electrode site; and P100 latency at FC3 was significantly earlier at FCz (M = 92.91, SD = 10.23) electrode site. P300 latency at FC3 (M = 235.44, SD = 4.57) electrode site was significantly earlier at FC4 (M = 262.56, SD = 8.57) electrode site. N400 latency at FC3 (M = 469.1, SD = 9.01) electrode site was significantly earlier at FC4 (M = 497.5, SD = 5.71) electrode site. However, there was no significant difference between group effects on LNP latency. There was a significant trial type main effect on LPP (Wilks' Lambda = .855 F (1, 38) = 6.445, $p < .05$, $\eta^2 = .15$) latency at posterior frontal electrode sites. Post hoc analysis showed that LPP latency in misleading trial (M = 665.2, SD = 13.8) electrode site was significantly earlier in latency than resolvable trial (M = 710.8, SD = 11.80) electrode site. There was interaction effect between location and trial type on latency (See Figure 5).

In general, results indicated that there was no significant difference between electrode sites and trial types in terms of groups. However, peaks had more prominent patterns than group differences. In probe-1 trials, amplitude of especially N100 and N400 peaks differences were more prominent in terms of trial type main effect and interaction effect at both anterior and posterior frontal electrode sites. In probe-2 trial type, especially amplitude and latency of P300, N400 and LNP peaks were affected up to location differences at posterior frontal electrode sites. According to the visual inspection results, N100, N400, P200 and P300 components were more prominent than the other peaks for both resolvable and misleading trials in probe-1 and probe-2 trial types. Although addicted produced higher P300 peaks than controls in misleading trials, results were not statistically significant.

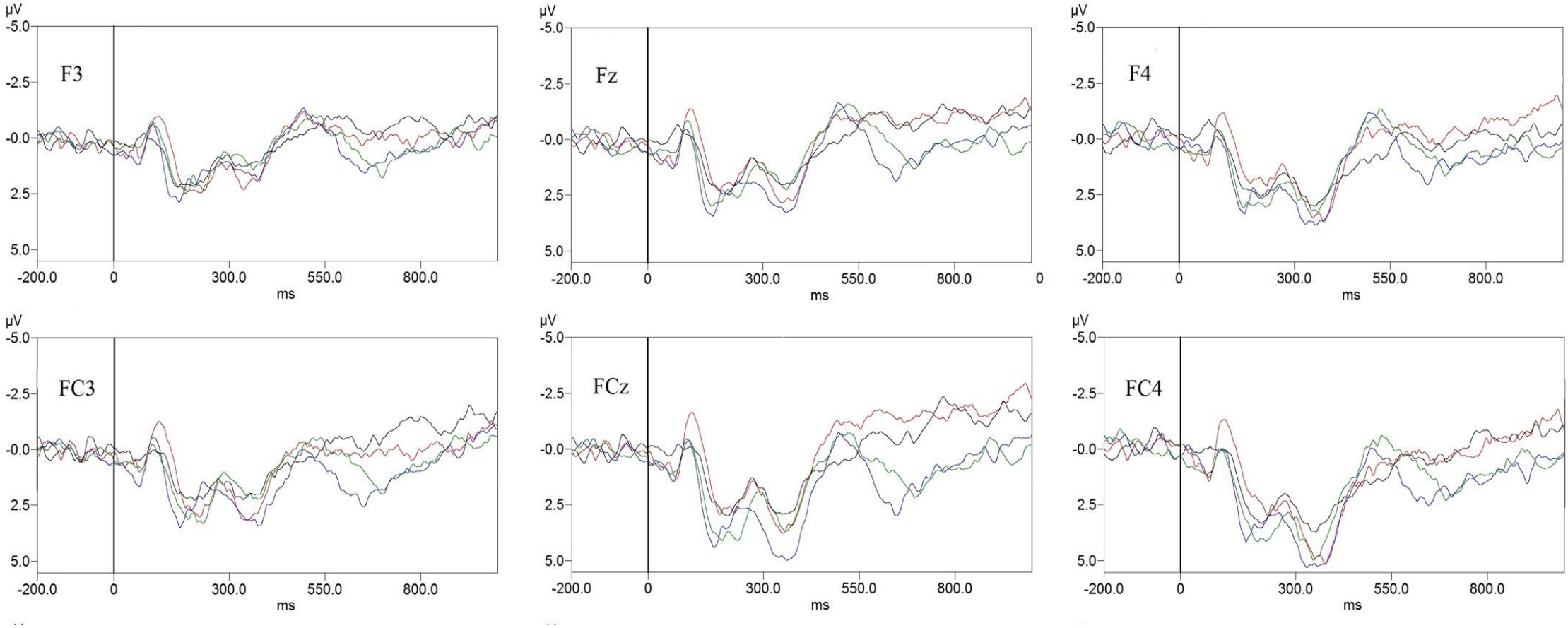

Figure 5. ERP grand average waveforms of 6 electrodes during Probe-2 Resolvable vs. Misleading Trials of Addicted and Control Groups

CHAPTER 4

DISCUSSION

4. 1. Overview

The aim of the study was to investigate the effects of violent video game addiction on ERPs under working memory. It is designed to investigate possible differences between excessive violent video game players and non-players in terms of their behavioral and electrophysiological performances during working memory task. In light of this aim, an adapted version of working memory task (Harkin & Kessler, 2009) was used and behavioral data and ERPs of participants were recorded during different phases of working memory task.

The statistical analysis was conducted for behavioral results and grand average stimulus-locked ERPs. During the analysis, the data were analyzed separately for resolvable and misleading conditions of probe-1 and probe-2 phases in line with the given task. The grand average analyses of stimulus-locked ERPs (amplitude and latency) were conducted separately for central, frontal, parietal and occipital electrode sides. Peaks of interest were analyzed for N100, P200, P300 and N400.

4. 1. 1. Summary of the Results. The present study tried to give an explanation for psychological and neural correlates of working memory under violent video game addiction effects. The behavioral results demonstrated that control group performed at resolvable and misleading condition of both phases (except resolvable in probe-1) in the task in terms of accuracy. On the other hand, addicted group was faster in giving responses in resolvable and misleading condition of both phases (except misleading in probe-1) in the task in terms of reaction time. However, there was only one statistically significant difference in misleading condition of probe-2 in which addicted group was slower in giving response compared to control group.

Stimulus-locked ERPs that were recorded while participants gave responses about the locations of previously encoded letters exposed the component represented at 100, 200, 300 and 400 ms time window (N100, P200, P300 and N400) as a result of mental evaluation. The N100 component was the first neural response after stimulus onset was and it was thought to be related with attentional activation at 100 ms time window. Second, the greater P200 component was recorded at parietal and occipital electrode sites for addicted group, whereas greater P200 component was recorded at frontal electrodes for control group. Detected P200 component was the highest for the control group and it was thought to be associated with the cognitive load of the task in misleading condition. Lastly, greater frontal P300 component was recorded for control group and it was linked to participants' cognitive demand for updating their memory assumptions.

Both results from behavioral and ERPs analysis were interpreted in light of what is comprehended from behavioral, neuroimaging and neuropsychological studies of the working memory literature.

4. 2. Literature Discussion for Behavioral Results

Impact of violent video game addiction on cognitive functions has been controversial in literature. Some studies (such as Boot et al. 2008; Coltazo et al. 2013; Ferguson, 2007; Green & Bavelier, 2003, 2015) claimed that violent video game has a positive effect on cognitive skills whereas other studies (such as Boot et al., 2008; Kuss & Griffith, 2012) showed declines in cognitive performances.

Violent video game playing requires monitoring and reacting quickly to many stimuli at the same time, and also selecting rapidly the relevant information to the given task and ignoring the irrelevant ones. Moreover, it requires avoiding unnecessary actions and quickly switching between tasks. So, playing violent video games are expected to enhance players' cognitive ability, especially working memory which requires monitoring and updating the coming information (Colzato et al., 2013), and also visuo-spatial working memory which includes remembering location and identifying of objects in the specific environment (Boot et al., 2008).

One of the main assumptions regarding the performance of working memory is that, playing video games might result in improvement in working memory ability of video game players, since players should collect and remember many stimuli or task at the same time in order to be successful in violent video games (Mahncke et al., 2006). Boot et. al (2008) conducted a study in which they trained the expert and non-expert game players, they found that expert video game players showed greater performances than non-expert group. In their following study (Boot et. al, 2010), the participants, who play video games less than 3 hr a week, were trained for a new nonviolent video game. They revealed that participants had greater performances on several memory including working memory and short-term memory after they were trained. Consistent with their

study, Colzato et al. (2013) detected improvement in working memory performance of video game players compared to non-game players.

Green and Bavelier (2003) found expert action video game players had higher performances than non-video game players in terms of visuospatial and attentional capacity. After they replicated and elaborated their findings using a training method in another study (Green & Bavelier, 2006 a, b), they found that non-video game player improved their skills in also objects tracking as video game players did. Thus, they suggested that changes in performances may be attributable to improvement in visual short-term memory skill. Their results were consistent with Boot et al.'s (2008), and Sungur and Boduroğlu's (2012) studies in which expert video game players showed superior visual short-term memory performances (Latham et al., 2013).

Our research, on the other hand, was designed to examine a specific cognitive skill in order to investigate any kinds of difference between working memory performances of game addicts and non-players. Thus, it was expected that addicted group have higher scores on accuracy and faster reaction time during working memory task. Second phase (probe-1) of the experiment was designed to reveal differences between addicted and control groups in terms of performances based on deciding the location of the given letter which could be part of original encoding set (resolvable trial) or not (misleading trial). Third phase (probe-2), on the other hand, was employed to see differences in terms of group performances based on deciding whether given letter was on the correct location as in original encoding set or not. However, the result of the analysis did not support our hypothesis.

According to the results, addicted group have more correct responses in probe-1 phase compared to control group but they have less correct responses in probe-2 phase. Since the probe-2 was harder phase than probe-1, this result showed that accuracy of the

responses of addicted group decreased when the task got harder. According to Harkin and Kessler (2009), misleading trial of probe-1 phase would interfere with working memory representations. Thus, when the participants repeatedly compare the probe letter given to the encoding set held in memory, this would result in a competition between a strong stimulus and weaker memorized letter-location bindings. Competition becomes strong when participants was unable to suppress the misleading information, original encoded memories get weaker which leads to a decrease in performance in probe-2. This effect could be supported by the reaction times, where addicted group performed worse and wasted more time to give a response. Although performances of both group decreased and reaction times got longer in probe-2 than probe -1, control group performed better than addicted group in probe-2.

Even though our study did not show a significant difference between addicted and control group in terms of accuracy, the results of the study indicated that the groups were significantly differed regarding reaction time for incorrect responses at probe-2 phase. Results showed that control group spent more time thinking before giving the responses than addicted group when the location of the given letter was wrong in probe-2. This result could be interpreted by the explanation by Castel et al. (2005) who reported that game-players displayed faster overall reaction times relative to non-players (Bailey & West, 2013).

In the study conducted by Littel et al. (2012), results revealed some similarities between substance dependence and impulse control disorders regarding reduced inhibition and high impulsivity in excessive game players compared to the control group via a Go/NoGo paradigm. They also stated that excessive game players produced decreased frontocentral ERN amplitudes with incorrect trials in comparison to correct trials which indicated poor error-processing. Since players are required to decide and act

quickly to be successful while playing, they have to sacrifice the right decisions and actions. Similarly, Dong et al. (2010) employed a study to investigate the response inhibition process of individuals with internet addiction by using a Go-NoGo task and revealed that internet addicts showed decrease in NoGo N200 amplitudes -which implies response inhibition- compared to controls (Kuss & Griffiths, 2012). They stated Additionally, video-game addicted did not show any significant increase in terms of correct answers compared to control group. These findings were contradictory to previous studies (e.g. Barlett et al., 2009; Boot et al., 2008; Colzato et al., 2013; McDermott et al., 2014) claiming enhancement or no changes (Irons et al, 2011; Powers et al., 2013) in working memory performance. However, it was taken into consideration that inhibition observed in reaction times of working memory task in our study was not the same as in Go/No-Go task.

Taken together, reason for the result of current study could be explained by high impulsivity of addicted which includes error processing and response inhibition insufficiencies (Kuss & Griffiths, 2012; Littel et al., 2012), and also decreased awareness and cognitive control in terms of errors (Irak, Soylu & Çapan, 2016). Addicted group had better performances regarding accuracy in probe-1, but their performances decreased when the task got harder compared to control group.

4. 3. Literature Discussion for ERPs Results

ERP results of the current study present both extensions of and contrasts to ERP studies that investigated respectively response monitoring, decision making and updating mechanisms. ERPs were recorded when participants were performing working memory task based on firstly giving response to where was the given letter (probe-1 phase) and then deciding whether the location was of the given letter was correct or not.

(probe-2 phase) Finding of the study is that working memory performances were related with the components recorded at 100, 200, 300 and 400 ms time window (N100, P200, P300 and N400 peaks, respectively) as a result of searching, recalling and recognition about encoding letters and their locations.

In general, visual inspection results showed that the N100 component was greater in amplitude for addicted group whereas the P200 and the P300 component were higher in amplitude for control group. Although the statistical analysis failed to show a significant difference between the addicted and control groups in terms of amplitude and latency values, P300 amplitude was reduced in addicted group as in accordance with the related literature.

The organization of the discussion part of the ERP results was divided into five sections that aimed to cover namely peaks of interest and strength and weakness of the study, and then future directions. The difference between addicted and control groups according to location of the electrodes on ERPs were examined for each component.

4. 3. 1. Interpretations of the results of N100 component. The visual N100 subcomponents come together to form N100. The earliest one peaks 100-150 ms after the stimulus at anterior electrode sites, whereas the posteriors arising from parietal cortex and lateral occipital cortex peaks 150-200 ms after the stimulus. The N100 components were shown to be influenced with spatial attention. Especially the lateral occipital N100 was detected to be greater in discrimination tasks than detection tasks. Thus, lateral occipital N100 was mainly associated with discriminative processing (Luck et al., 2000). On the other hand, in current study, N100 component was recorded within 80- 150 ms (anterior dominant) time window as the first neural response following the presence of stimulus. This occurrence may indicate a selection process for

upcoming stimuli. Moreover, it could be accepted as a primary stage of further processes for attended stimuli (Hillyard & Anllo-Vento, 1998; Hillyard & Picton, 1979; Luck et al., 1990).

Visual analysis of current study revealed that addicted group's amplitude of N100 was higher than control group at frontal electrode sites in probe-1 phase. Additionally, addicted group also elicited higher N100 amplitudes than control group in both resolvable and misleading trials of probe-2 phase. The results can be explained by improved attention skills of violent video game addicted group. Hence, it can be perceived to reflect of perceptual preparation or pre-attentive process for the activation in this study.

According to Blacker et al. (2014), previous studies with training strategies showed that participants improving their visual working memory skill after training were seem to use their orienting attention more proficiently than individuals with lower visual working memory capacity by spatially positioning their attention to groups of objects or locations to be encoded into visual working memory. Since action video game players were required to be interacted with complex visual environments in games, their visual cognitive skills such as spatial distribution of attention (Green & Bavelier, 2003, 2006a), temporal resolution of attention (Green & Bavelier, 2003), selective attention (Bavelier, Achtman, Mani, & Focker, 2012; Karle, Watter, & Shedden, 2010) and visual speed of processing (Dye, Green, & Bavelier, 2009) were improved (as cited in Blacker et al., 2014). Consistently, Sungur and Boduroğlu (2012) also found in their study that action video game players performed better at representing memory items with greater precision in a delayed localization task than non-players. According to N100 component literature, N100 component was associated with working memory in terms of directing attention into meaningful sensory information

and blocking unnecessary stimuli (Annanmaki et al., 2017; Luck et al., 1990). However, the latency of N100 was prolonged for addicted group than control group in resolvable trials. It can be interpreted in such way that control group had greater sensory processing skills, so they were quicker at stimulus detection compared to addicted group.

McEvoy et al. (2001) explained the delay in N100 component as a constant feature within levels of memory load, and within the matching / non-matching aspect of the stimulus. Thus, it proposed a reflection of slowing in perceptual processing speed. Although the latency of N100 was prolonged in addicted group, their amplitude was not affected. According to McEvoy et al. (2001), when the stimuli come across the visually attended area, early-latency of visual ERPs are enriched. Then, reaction times also get shorter to stimuli presented. In light of this information, the explanation could be the selective attention to letter-location matching with the encoded set held in memory which, in turn, caused an increased N100 amplitude of addicted group in resolvable trials in the present study. Therefore, it may result in differences in reaction times recorded between addicted and control groups.

Taken together, our results were partially in consistency with previous studies suggested that N100 amplitude was increased in game-addicted group. Consequently, addiction might cause a slowdown in sensory processing, whereas it does not affect the attention skills.

4. 3. 2. Interpretations of the results of P200 component. The amplitude of P200 component of ERP was associated with early visual stimuli perception (Wu et al., 2012) and mainly generated in parieto-occipital regions. P200 component was seen as

an indicator of working memory function, particularly encoding (Finnigan, O'Connel & Robertson, 2010).

Finnigan, O'Connel and Robertson (2010) mentioned that in studies exploring age-related cognitive decline via working memory task, it was found that young adults elicited higher P200 amplitudes than older ones. Especially, in a study employed with a modified Sternberg paradigm, the significant correlation was found between P200 latency and task performance. Since no age effect was detected for recognition accuracy and a relation with P200 latency, results were attributed to task conditions including higher cognitive load. The authors claimed that decline in attention and encoding could lead working memory function to decrease in healthy older adults. Within this context, a later P200 component may be associated with more inclusive amount of encoding-related activity contribute to better memory performance in general (Finnigan, O'Connel and Robertson, 2010).

Our results could be interpreted in a parallel way with the previous studies discussed above. According to our results, control group produced bigger P200 amplitudes at frontal central electrode sides than addicted group in terms of misleading trial of both probe-1 and probe-2 phases, although addicted group produced higher amplitudes than control group in both conditions at occipital and parietal electrode sites. In misleading trials, group differences were more observable in terms of P200 components at frontal electrode sites.

As aforementioned, P200 component was dominantly elicited in occipital and parietal which also comprised of visual dorsal stream. Visual dorsal stream is associated with the visual spatial location (Ungerleider et al., 1998). Thus, addicted group had greater P200 amplitude in these brain regions which meant that they were better at

encoding the location of resolvable probe letter compared to control group. However, their P200 amplitude declined at frontal electrode sites when the cognitive load increased in misleading trial. Since working memory performance had frontal lobe dominance (Courtney et. al, 1998; Nissim et. al, 2017), the explanation could be that visuospatial working memory performances of addicted group decreased with task demanding. The latency differences of P200 components between groups were clearly observable in misleading trials in which the latency of P200 was earlier for control group than addicted group. As aforementioned, this same latency pattern was also observed for N100 component. This result suggested that control group had greater encoding process compared to addicted group, thus they were faster to search for probe letter in the original encoding set held in working memory.

In the light of the related literature discussed above, our results were in accordance with previous studies and suggested that performance of addicted group decreased when cognitive load increased in misleading trials in terms of P200 amplitude.

4. 3. 3. Interpretations of the results of P300 component. The P300 is seen as an indicator of human attention, feeling, memory updating, and decision, and is derived from parietal and occipital lobes during information processing (Liu et al., 2015). The P300 is the most studied component for ERP studies, especially in working memory research. It was found that amplitude of P300 component of the ERP has been related to (updating of) working memory (Donchin & Coles, 1988; Kramer et al., 1986; Polich & Kok, 1995). In previous studies on violent video game addiction conducting with ERP, results showed decrease in amplitude and increase in latency of P300 component (Irak, Soylu & Çapan, 2016; Kuss & Griffiths, 2012) Yu et al. (2009)'s study revealed that

excessive internet use resulted in reduced P300 amplitude and increased P300 latency. Reduced P300 amplitude and increased P300 latency were detected also in people with substance dependency (Ge et al., 2011). P300 amplitude was also linked to evaluative decision-making. According to this, the more a stimulus engages a relevant motivational system, the larger the P300 amplitude (Engelhardt et al., 2011).

In the present study, it is aimed to examine if the violent game addiction had an effect on P300 component that amplitude and latency values of game-addicted differ comparing to non-players. Results showed that P300 components at posterior frontal electrode sides were higher in amplitude than at anterior frontal sides in probe-1 phase which included resolvable and misleading trials. Amplitude values of addicted group in resolvable trial were higher than misleading trial in terms of P300 components. According to these results, it could be claimed that when the cognitive load gets bigger, amplitude of P300 in addicted group decreases. This explanation could be used for the results of probe- 2 phase. In probe-2 phase, it was recorded that addicted group produced greater P300 amplitude than control group in resolvable trials at frontal electrode sites. On the other hand, in misleading trials, control group produced higher P300 amplitude than addicted group at frontal electrode sites. In the light of these results, it can be stated that our results were consistent with the previous studies (Boot et al., 2008).

According to Watter et al. (2001), influential triarchic model of Johnson developed in 1986 and 1993, indicates that when the complexity of task and stimulus, value of stimulus and overall information transmitted to the participant are greater, in a combination with lower stimulus probability and sequential expectation of stimuli, they all lead P300 amplitudes to be greater. Additionally, they claimed that P300 amplitude also rises proportionately with allocated processing capacity is greater. In dual-task

paradigms such as Sternberg paradigm developed in 1966, the P300 amplitude arising from a secondary task also gets smaller when the primary task gets harder. It points out that reallocation of processing capacity goes away from the secondary task to the primary task. Another explanation for decrease in P300 amplitude of addicted group could be the speed of stimulus identification. Yu et al. (2009) found that participants with internet addiction disorder in their study produced smaller P300 amplitude and longer P300 latency. They interpreted the results in such a way that either participants with internet addiction disorder did not do not have enough attention resources or allocate attention resources improperly (Kuss & Griffiths, 2012; Yu et al., 2009). Likewise, the speed of stimulus identification was reduced in the participants with internet addiction disorder, so P300 latency was longer.

Thus, our results showing decline in P300 amplitude when the task got harder, could be explained and supported with these theories. Addicted group outperformed control group in resolvable trials but had lower performances when task was more demanding.

Since latency of P300 was seen to be influenced by perceptual complexity and cognitive processing demands of a given task (Strayer, Wickens & Braune, 1987), and also associated with memory search and stimulus category decision/evaluation processes (Strayer, Wickens & Braune, 1987; Kramer et al., 1991), studies examining Sternberg paradigm found that increase in the latency of P300 was directly proportionate with increase in memory set. According to these studies, serial search for items held in memory was exhaustive so both reaction time and P300 latency increased with memory load gets bigger (Watter et al., 2001). Moreover, Ge et al. (2011) explained the prolonged P300 latency of participants with internet addiction disorder, as such an index of neurodegenerative processes which had an influence on callosal size

and the efficiency of interhemispheric transmission. Therefore, they concluded that people with internet addiction disorder could have difficulties in perception speed. In the light of this information, it was not surprising to see that P300 latency of addicted group increased in misleading trial of our study. Taken together, the explanation could be that because participants were forced to determine if the given letter was a match or mismatch with the original encoding set held in memory before deciding the exact location of the letter.

4. 3. 4. Interpretations of the results of N400 component. The N400 is seen as a dependent measure of different cognitive processes such as language processing, object, face, action, and gesture processing, mathematical cognition, semantic and recognition memory (Kutas & Federmeier, 2011). It was recorded as the largest in presence of semantic anomalies, but also present for improbable but sensible endings. N400 is a relative negativity peaking around 400 ms after stimulus presentation which can be seen as a reflection of lexical-semantic processing. Neural N400 components were elicited in prefrontal cortex (PFC) and inferior frontal cortex. Especially, if the stimuli are nonlinguistic but meaningful. To illustrate, a line drawing produces N400, when there is inconsistency between the semantic context generated and previous word sequence or line drawings. (Luck, 2014, p. 104). Thus, amplitude of N400 decreases when the target word becomes more predictable which makes it easier to integrate into the context (Steinhauer et al., 2017).

According to Kutas, van Petter and Kluender, N400 amplitude is related with stored representations of a specific stimulus and retrieval clues provided by the previous context. If a stored conceptual knowledge related with a word or other meaningful stimuli is easy or difficult, amplitude of N400 changes. On the other hand, Hagoort

suggested another theory for N400, which claimed that N400 is elicited with word meaning retrieved is integrated into previous discourse. When the work load needed to maintain this integration, N400 amplitude becomes larger (Luck, 2014, p. 105).

Results of the current study demonstrated that there was a significant difference between the location and trial type of frontal electrode regions in probe-1. Groups showed greater N400 amplitudes in misleading trials than resolvable trials which could be expected in accordance with the information above. Specifically, control group produced greater N400 amplitudes than addicted group in both resolvable and misleading trials. Since misleading letter interfered with the semantic context previously held in working memory, it was not surprising to observe larger N400 amplitudes in these trials. In the light of the Hagoort's theory suggesting that work load for integration got larger because individuals tried to find, activate the meaning of the word and integrate it to the stored information, results of our study could be interpreted as such that control group were better at processing this integration compared to addicted group.

4. 4. Limitations and strengths of the current study, and recommendations for the future research

On the basis of related literature, there are just several EEG/ERP studies on violent video game addition. Additionally, many studies focused on many cognitive functions at the same time. The current study addresses the neural correlates of working memory performance in addiction using ERPs. Investigating the relationship between behavioral data and ERP data is valuable for the understanding of implicit expressions of knowledge. Many of our findings were in accordance with addiction and working memory literature.

There were inconsistent findings in the related literature about the effects of video game addiction on working memory performance. Our results contributed to some of them claiming that violent game addiction has negative consequences on working memory performance. Also, it was recorded that game addicted group could have been difficulties with updating working memory and searching for encoded items held during working memory process, especially when task got more demanding. They also had higher impulsiveness which led them to make sometimes incorrect responses. On the other hand, they were seemed to have improved attention skills and speed of reaction.

Despite given the paucity of the present study, there are some shortcomings that future studies should take into consideration in order to improve the understanding about working memory performances in relation with violent video game addiction. Considering our results, there are several issues needed to be discussed in order to realize the shortcomings of the present study.

First of all, methods using to measure the working memory performance can be seen as a limitation of our study. One possible explanation for the differences of our results from the previous studies could be related to different methods conducted by the studies. Previous studies which revealed significant effects of playing video games on working memory applied mainly a practice or training strategy (e.g., Barlett et al., 2009; Basak et al., 2008; Boot et al., 2008). In current study, addicted and control groups were categorized based on the self-report of the individuals about their approximate amount of time spent on playing violent video games per week. Similarly, Iron et al (2011) also included participants who had been playing violent video games between 4 and 20 hours a week. Neither training nor practice strategies were used in their study. Results of their study revealed no differences skills between players and non-players in terms of

cognitive skills. Thus, it would be better for future studies to control duration of training and time spent for playing video games in experimental laboratory studies.

Additionally, another limitation could be the experience of namely game addicted participants. We did not take into consideration how long individuals engage in violent video games. In our sample, it was possible that some individuals have been playing for many years while others have been playing for a short period of time. In the study conducted by Colzato et al. (2013), participants who played video games at least 5 hours in a week for minimum period of 1 year were included. Working memory performances were measured with an n-back task. Results showed that violent video game players outperformed non-players. In current study, differences between experiences of violent video game players might impact our results. Thus, differences between game experiences of groups may result in playing performances. It would be better for future studies to divide namely addicted group based on their experience level.

The working memory task employed could be another reason why groups did not significantly differ from each other. Working memory task in our study was different from the previous studies and it was adapted from Harking and Kessler's task. Generally, an n-back task was used in previous studies. Harkin and Kessler (2009) claimed that the task conducted in their research was very easy which made it difficult to reveal group differences. Thus, it would be better to employ a different but harder task to reveal group differences or two different tasks could be used with counterbalancing to eliminate the task factor in the future studies.

Finally, "violent" term could be another limitation for our study. "Violent" definition is relative and difficult to describe. In current study, participants were asked to score the violence level of the games they played. According to their self-ratings,

some games were just action games and included no harmful actions such as LoL. However, in psychological perspective, a gun vision could be seen as violent content in video games. Thus, it is unclear if the violent content had really impact on working memory performance observed. It could be better to clearly define “violent” term and games should be categorized more cautiously in terms of their content.

REFERENCES

Annanmaki, T., Palmu, K., Murros, K., & Partanen, J. (2017). Altered N100-potential associates with working memory impairment in Parkinson's disease. *Journal of Neural Transmission*, *124*(10), 1197-1203.

Arriaga, P., Adrião, J., Madeira, F., Cavaleiro, I., Maia e Silva, A., Barahona, I., & Esteves, F. (2015). A "dry eye" for victims of violence: Effects of playing a violent video game on pupillary dilation to victims and on aggressive behavior. *Psychology of Violence*, *5*(2), 199.

Baddeley, A. D., & Hitch, G. (1974). Working memory. In *Psychology of learning and motivation* (Vol. 8, pp. 47-89). Academic press.

Baddeley, A. (2000). The episodic buffer: a new component of working memory?. *Trends in cognitive sciences*, *4*(11), 417-423.

Bailey, K., & West, R. (2013). The effects of an action video game on visual and affective information processing. *Brain research*, *1504*, 35-46.

Barlett, C. P., Anderson, C. A., & Swing, E. L. (2009). Video game effects—confirmed, suspected, and speculative: a review of the evidence. *Simulation & Gaming*, *40*(3), 377-403.

Bartholow, B. D., Bushman, B. J., & Sestir, M. A. (2006). Chronic violent video game exposure and desensitization to violence: Behavioral and event-related brain potential data. *Journal of experimental social psychology*, *42*(4), 532-539.

Bartholow, B. D., Sestir, M. A., & Davis, E. B. (2005). Correlates and consequences of exposure to video game violence: Hostile personality, empathy, and aggressive behavior. *Personality and Social Psychology Bulletin*, *31*(11), 1573-1586.

Basak, C., Boot, W. R., Voss, M. W., & Kramer, A. F. (2008). Can training in a real-time strategy video game attenuate cognitive decline in older adults?. *Psychology and aging*, *23*(4), 765.

Blacker, K. J., Curby, K. M., Klobusicky, E., & Chein, J. M. (2014). Effects of action video game training on visual working memory. *Journal of Experimental Psychology: Human Perception and Performance*, *40*(5), 1992.

Block, J. J. (2008). Issues for DSM-V: Internet addiction. *American journal of Psychiatry*, *165*(3), 306-307.

Boot, W. R., Kramer, A. F., Simons, D. J., Fabiani, M., & Gratton, G. (2008). The effects of video game playing on attention, memory, and executive control. *Acta psychologica*, *129*(3), 387-398.

Boot, W. R., Basak, C., Erickson, K. I., Neider, M., Simons, D. J., Fabiani, M., ... & Low, K. A. (2010). Transfer of skill engendered by complex task training under conditions of variable priority. *Acta psychologica*, *135*(3), 349-357.

Colzato, L. S., Van Leeuwen, P. J., Van Den Wildenberg, W., & Hommel, B. (2010). DOOM'd to switch: superior cognitive flexibility in players of first person shooter games. *Frontiers in psychology*, *1*, 8.

Colzato, L. S., van den Wildenberg, W. P., Zmigrod, S., & Hommel, B. (2013). Action video gaming and cognitive control: playing first person shooter

games is associated with improvement in working memory but not action inhibition. *Psychological research*, *77*(2), 234-239.

Courtney, S. M., Petit, L., Maisog, J. M., Ungerleider, L. G., & Haxby, J. V. (1998). An area specialized for spatial working memory in human frontal cortex. *Science*, *279*(5355), 1347-1351.

Donchin, E., & Coles, M. G. (1988). Is the P300 component a manifestation of context updating. *Behavioral and brain sciences*, *11*(3), 357-427.

Elmore, W. R. (2012). *The effect of violent video game play on emotion modulation of startle*. University of Missouri-Kansas City.

Elson, M., & Ferguson, C. J. (2014). Does doing media violence research make one aggressive?. *European Psychologist*.

Ferguson, C. J. (2007). The good, the bad and the ugly: A meta-analytic review of positive and negative effects of violent video games. *Psychiatric Quarterly*, *78*(4), 309-316.

Finnigan, S., O'Connell, R. G., Cummins, T. D., Broughton, M., & Robertson, I. H. (2011). ERP measures indicate both attention and working memory encoding decrements in aging. *Psychophysiology*, *48*(5), 601-611.

Ge, L., Ge, X., Xu, Y., Zhang, K., Zhao, J., & Kong, X. (2011). P300 change and cognitive behavioral therapy in subjects with Internet addiction disordera 3-month follow-up study. *Neural Regeneration Research*, *6*(26), 2037-2041.

Gentile, D. A., Swing, E. L., Anderson, C. A., Rinker, D., & Thomas, K. M. (2016). Differential neural recruitment during violent video game play in violent-and nonviolent-game players. *Psychology of Popular Media Culture*, *5*(1), 39.

Gonnerman Jr, M. E., & Lutz, G. M. (2011). *Gambling attitudes and behaviors: A 2011 survey of adult Iowans*. Center for Social and Behavioral Research, University of Northern Iowa.

Green, C. S., & Bavelier, D. (2003). Action video game modifies visual selective attention. *Nature*, *423*(6939), 534.

Green, C. S., & Bavelier, D. (2015). Action video game training for cognitive enhancement. *Current Opinion in Behavioral Sciences*, *4*, 103-108.

Green, C. S., & Seitz, A. R. (2015). The impacts of video games on cognition (and how the government can guide the industry). *Policy Insights from the Behavioral and Brain Sciences*, *2*(1), 101-110.

Griffiths, M. (1999). Violent video games and aggression: A review of the literature. *Aggression and violent behavior*, *4*(2), 203-212.

Griffiths, M. (2000). Internet addiction-time to be taken seriously?. *Addiction research*, *8*(5), 413-418.

Hambrick, D. Z., Oswald, F. L., Darowski, E. S., Rench, T. A., & Brou, R. (2010). Predictors of multitasking performance in a synthetic work paradigm. *Applied cognitive psychology*, *24*(8), 1149-1167.

Han DH, Bolo N, Daniels MA, Arenella L, Lyoo IK, Renshaw PF. Brain activity and desire for internet video game play. *Comprehensive psychiatry*. 2011;52(1):88-95. doi:10.1016/j.comppsych.2010.04.004.

Harkin, B., & Kessler, K. (2009). How checking breeds doubt: Reduced performance in a simple working memory task. *Behaviour Research and Therapy*, *47*(6), 504-512.

Hillyard, S. A. & Picton, T. W. (1979). Event-related brain potentials and selctive information processing in man. *Progress in Clinical Neurophysiology*, 6, 1-50.

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. *Proceedings of the National Academy of Sciences*, *95*(3), 781-787.

Irak, M., Soylu, C., & Çapan, D. (2016). Violent Video Games and Cognitive Processes: A Neuropsychological Approach. In *Gamer Psychology and Behavior* (pp. 3-20). Springer International Publishing.

Irons, J. L., Remington, R. W., & McLean, J. P. (2011). Not so fast: Rethinking the effects of action video games on attentional capacity. *Australian Journal of Psychology*, *63*(4), 224-231.

Kensinger, E. A., & Corkin, S. (2003). Effect of negative emotional content on working memory and long-term memory. *Emotion*, *3*(4), 378.

Kirsh, S. J., Olczak, P. V., & Mounts, J. R. (2005). Violent video games induce an affect processing bias. *Media Psychology*, *7*(3), 239-250.

Kramer, A., Schneider, W., Fisk, A., & Donchin, E. (1986). The Effects of Practice and Task Structure on Components of the Event-Related Brain Potential. *Psychophysiology*, *23*(1), 33-47.

Kramer, A. F., Strayer, D. L., & Buckley, J. (1991). Task versus component consistency in the development of automatic processing: A psychophysiological assessment. *Psychophysiology*, *28*(4), 425-437.

Kuss, D. J., & Griffiths, M. D. (2012). Internet and gaming addiction: a systematic literature review of neuroimaging studies. *Brain sciences*, *2*(3), 347-374.

Kuss, D. J. (2013). Internet gaming addiction: current perspectives. *Psychology research and behavior management*, *6*, 125.

Kutas, M., & Federmeier, K. D. (2011). Thirty years and counting: finding meaning in the N400 component of the event-related brain potential (ERP). *Annual review of psychology*, *62*, 621-647.

Latham, A. J., Patston, L. L., & Tippett, L. J. (2013). The virtual brain: 30 years of video-game play and cognitive abilities. *Frontiers in psychology*, *4*, 629.

Lemmens, J. S., Valkenburg, P. M., & Peter, J. (2009). Development and validation of a game addiction scale for adolescents. *Media Psychology*, *12*(1), 77-95.

Lemos, I. L., Abreu, C. N. D., & Sougey, E. B. (2014). Internet and video game addictions: a cognitive behavioral approach. *Archives of Clinical Psychiatry (São Paulo)*, *41*(3), 82-88.

Lim, S., & Reeves, B. (2009). Being in the game: Effects of avatar choice and point of view on psychophysiological responses during play. *Media Psychology*, *12*(4), 348-370.

Littel, M., Berg, I., Luijten, M., Rooij, A. J., Keemink, L., & Franken, I. H. (2012). Error processing and response inhibition in excessive computer game players: an event-related potential study. *Addiction biology*, *17*(5), 934-947.

Liu, Z., & Guo, C. (2007). Interactions between working memory and selective attention. *Chinese Science Bulletin*, *52*(20), 2811-2819.

Liu, Y., Teng, Z., Lan, H., Zhang, X., & Yao, D. (2015). Short-term effects of prosocial video games on aggression: an event-related potential study. *Frontiers in Behavioral Neuroscience*, *9*, 193. http://doi.org/10.3389/fnbeh.2015.00193

Luck, S. J. An Introduction to the Event-Related Potential Technique.

Luck, S. J., Heinze, H. J., Mangun, G. R., & Hillyard, S. A. (1990). Visual event-related potentials index focused attention within bilateral stimulus arrays. II.

Functional dissociation of P1 and N1 components. *Electroencephalography and clinical neurophysiology*, *75*(6), 528-542.

Luck, S.J., Woodman, G.F., & Vogel, E.K. (2000). Event-Related potential studies of attention. *Trends in Cognitive Sciences*, 4, 432-440.

Mahncke, H. W., Connor, B. B., Appelman, J., Ahsanuddin, O. N., Hardy, J. L., Wood, R. A., ... & Merzenich, M. M. (2006). Memory enhancement in healthy older adults using a brain plasticity-based training program: a randomized, controlled study. *Proceedings of the National Academy of Sciences*, *103*(33), 12523-12528.

Mathews, V. P., Kronenberger, W. G., Wang, Y., Lurito, J. T., Lowe, M. J., & Dunn, D. W. (2005). Media violence exposure and frontal lobe activation measured by functional magnetic resonance imaging in aggressive and nonaggressive adolescents. *Journal of Computer Assisted Tomography*, *29*(3), 287-292.

Mathiak, K. A., Klasen, M., Weber, R., Ackermann, H., Shergill, S. S., & Mathiak, K. (2011). Reward system and temporal pole contributions to affective evaluation during a first person shooter video game. *BMC neuroscience*, *12*(1), 66.

McDermott, A. F., Bavelier, D., & Green, C. S. (2014). Memory abilities in action video game players. *Computers in Human Behavior*, *34*, 69-78.

McEvoy, L. K., Pellouchoud, E., Smith, M. E., & Gevins, A. (2001). Neurophysiological signals of working memory in normal aging. *Cognitive Brain Research*, *11*(3), 363-376.

Mehroof, M., & Griffiths, M. D. (2010). Online gaming addiction: the role of sensation seeking, self-control, neuroticism, aggression, state anxiety, and

trait anxiety. *Cyberpsychology, behavior, and social networking*, *13*(3), 313-316.

Nissim, N. R., O'Shea, A. M., Bryant, V., Porges, E. C., Cohen, R., & Woods, A. J. (2017). Frontal structural neural correlates of working memory performance in older adults. *Frontiers in aging neuroscience*, *8*, 328.

Polich, J., & Kok, A. (1995). Cognitive and biological determinants of P300: an integrative review. *Biological psychology*, *41*(2), 103-146.

Powers, K. L., Brooks, P. J., Aldrich, N. J., Palladino, M. A., & Alfieri, L. (2013). Effects of video-game play on information processing: a meta-analytic investigation. *Psychonomic bulletin & review*, *20*(6), 1055-1079.

Regenbogen, C., Herrmann, M., & Fehr, T. (2010). The neural processing of voluntary completed, real and virtual violent and nonviolent computer game scenarios displaying predefined actions in gamers and nongamers. *Social neuroscience*, *5*(2), 221-240.

Schröger, E., Mecklinger, A., & Pollmann, S. (2004). Special issue: working memory and cognition.

Shams, T. A., Foussias, G., Zawadzki, J. A., Marshe, V. S., Siddiqui, I., Müller, D. J., & Wong, A. H. (2015). The effects of video games on cognition and brain structure: potential implications for neuropsychiatric disorders. *Current psychiatry reports*, *17*(9), 71.

Steinhauer, K., Royle, P., Drury, J. E., & Fromont, L. A. (2017). The priming of priming: Evidence that the N400 reflects context-dependent post-retrieval word integration in working memory. *Neuroscience letters*, *651*, 192-197.

Strayer, D. L., Wickens, C. D., & Braune, R. (1987). Adult age differences in the speed and capacity of information processing: II. An electrophysiological approach. *Psychology and aging*, *2*(2), 99.

Sungur, H., & Boduroglu, A. (2012). Action video game players form more detailed representation of objects. *Acta Psychologica*, *139*(2), 327-334.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics*. Allyn & Bacon/Pearson Education.

Takeuchi, H., Taki, Y., Hashizume, H., Asano, K., Asano, M., Sassa, Y., ... & Kawashima, R. (2016). Impact of videogame play on the brain's microstructural properties: cross-sectional and longitudinal analyses. *Molecular psychiatry*, *21*(12), 1781.

Ungerleider, L. G., Courtney, S. M., & Haxby, J. V. (1998). A neural system for human visual working memory. *Proceedings of the National Academy of Sciences*, *95*(3), 883-890.

Wang, Y., Mathews, V. P., Kalnin, A. J., Mosier, K. M., Dunn, D. W., Saykin, A. J., & Kronenberger, W. G. (2009). Short term exposure to a violent video game induces changes in frontolimbic circuitry in adolescents. *Brain Imaging and Behavior*, *3*(1), 38-50.

Watter, S., Geffen, G. M., & Geffen, L. B. (2001). The n-back as a dual-task: P300 morphology under divided attention. *Psychophysiology*, *38*(6), 998-1003.

Weber, R., Ritterfeld, U., & Mathiak, K. (2006). Does playing violent video games induce aggression? Empirical evidence of a functional magnetic resonance imaging study. *Media psychology*, *8*(1), 39-60.

Wells, A. (2002). *Emotional disorders and metacognition: Innovative cognitive therapy*. John Wiley & Sons.

Wu, S., Cheng, C. K., Feng, J., D'angelo, L., Alain, C., & Spence, I. (2012). Playing a first-person shooter video game induces neuroplastic change. *Journal of cognitive neuroscience, 24*(6), 1286-1293.

Yu, H., Zhao, X., Li, N., Wang, M., & Zhou, P. (2009). Effect of excessive Internet use on the time–frequency characteristic of EEG. *Progress in Natural Science, 19*(10), 1383-1387.

APPENDIX A

Mean and SD of amplitude values for peaks of interest at 6 electrode locations

Electrode	Component	Probe-1/Resolvable			Probe-1/Misleading			Probe-2/Resolvable			Probe-2/Misleading		
		N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*
F3	N100	40	-2.64	1.98	40	-1.83	2.41	40	-1.70	2.46	40	-1.41	3.04
	N400	40	-3.51	3.77	40	-3.92	2.74	40	-2.56	3.78	40	-2.79	4.27
	P100	40	1.13	1.87	40	1.27	1.83	40	1.51	2.11	40	1.60	2.09
	P200	40	1.82	2.18	40	2.01	2.84	40	3.24	2.63	40	3.68	2.29
	P300	40	2.1	2.72	40	2.19	2.3	40	3.62	3.11	40	3.68	3.00
	LNP	40	-3.34	4.13	40	-2.4	3.49	40	-2.38	4.43	40	-1.17	4.44
	LPP	40	1.27	3.73	40	2.39	3.17	40	2.22	3.96	40	3.27	3.72
F4	N100	40	-2.87	2.60	40	-2.33	2.19	40	-1.93	2.29	40	-1.47	2.64
	N400	40	-2.62	3.69	40	-4.78	3.53	40	-1.33	3.39	40	-2.62	3.95
	P100	40	.75	2.5	40	.62	2.09	40	1.45	2.7	40	1.68	2.16
	P200	40	1.66	2.36	40	1.77	2.63	40	3.54	2.91	40	4.18	2.96
	P300	40	1.69	2.95	40	1.51	3.06	40	4.27	3.19	40	4.65	3.35

	LNP	40	-2.5	3.95	40	-3.57	3.73	40	-1.55	3.82	40	-.77	4.60
	LPP	40	2.00	3.9	40	1.15	2.97	40	2.72	3.63	40	3.97	3.51
Fz	N100	40	-3.26	2.44	40	-1.75	2.98	40	-2.11	2.76	40	-1.35	3.68
	N400	40	-3.45	3.43	40	-4.38	3.7	40	-2.37	3.77	40	-2.79	4.97
	P100	40	.86	2.30	40	.95	2.11	40	1.36	2.59	40	1.52	2.27
	P200	40	1.74	2.15	40	2.23	2.86	40	3.28	3.28	40	4.69	3.06
	P300	40	1.82	3.03	40	2.03	3.29	40	3.78	3.51	40	4.74	3.62
	LNP	40	-3.88	3.71	40	-3.03	3.96	40	-3.04	4.00	40	-1.55	4.96
	LPP	40	1.04	3.86	40	1.89	3.24	40	1.54	3.52	40	3.76	4.02
FC3	N100	37	-2.77	2.67	37	-1.69	2.42	40	-1.89	2.81	40	-1.1	3.14
	N400	37	-4.13	4.33	37	-4.66	4.02	40	-1.71	4.18	40	-1.33	4.9
	P100	37	1.49	2.39	37	1.61	2.23	40	2.12	2.4	40	2.26	2.05
	P200	37	1.69	2.31	37	1.96	2.95	40	3.48	3.03	40	4.57	3.1
	P300	37	2.08	3.22	37	1.9	3.42	40	4.54	3.38	40	4.81	3.53
	LNP	37	-4.17	4.52	37	-2.31	3.59	40	-2.33	4.23	40	-.34	4.88
	LPP	37	.71	4.38	37	2.31	3.78	40	2.61	3.66	40	4.42	4.03
	N100	37	-2.68	2.53	37	-1.79	2.36	40	-1.71	3.11	40	-.92	3.14

	N400	37	-2.43	3.47	37	-4.75	3.83	40	-.61	3.69	40	-1.77	4.04
	P100	37	1.49	2.29	37	1.41	1.96	40	2.29	3.00	40	2.77	2.75
	P200	37	2.08	2.57	37	2.15	2.68	40	4.14	3.22	40	5.22	3.16
FC4	P300	37	2.23	3.47	37	2.20	3.02	40	5.34	3.61	40	6.01	4.23
	LNP	37	-2.63	3.73	37	-3.58	3.32	40	-1.62	3.74	40	-1.04	4.42
	LPP	37	2.07	3.53	37	1.71	3.14	40	2.99	3.89	40	4.3	4.2
	N100	37	-2.86	2.79	37	-1.80	2.86	40	-2.46	2.91	40	-1.92	3.3
	N400	37	-3.78	3.82	37	-5.13	3.76	40	-2.65	5.14	40	-2.42	4.60
	P100	37	1.62	2.62	37	1.64	2.37	40	1.96	2.91	40	1.90	2.75
FCz	P200	37	2.41	2.67	37	2.54	3.3	40	4.02	3.47	40	5.44	4.21
	P300	37	2.28	4.16	37	2.28	3.89	40	4.83	4.19	40	5.95	4.70
	LNP	37	-4.09	4.48	37	-3.31	3.93	40	-3.71	5.38	40	-1.3	5.01
	LPP	37	1.27	4.44	37	2.26	3.51	40	1.62	5.1	40	4.56	4.63

APPENDIX B

Mean and SD of latency values for peaks of interest at 6 electrode locations

Electrode	Component	Probe-1/Resolvable			Probe-1/Misleading			Probe-2/Resolvable			Probe-2/Misleading		
		N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*
F3	N100	40	117.33	22.85	40	113.30	22.98	40	107.28	18.77	40	99.78	18.21
	N400	40	447.18	71.42	40	429.00	92.91	40	475.15	80.88	40	448.20	122.41
	P100	40	68.73	15.53	40	70.13	15.97	40	70.43	17.24	40	71.13	16.68
	P200	40	174.83	24.02	40	166.75	28.05	40	174.28	22.73	40	161.00	33.13
	P300	40	242.85	31.51	40	239.40	38.44	40	231.73	38.59	40	227.93	56.04
	LNP	40	686.53	42.59	40	651.40	105.81	40	651.25	107.11	40	629.45	172.74
	LPP	40	734.45	93.09	40	727.60	133.52	40	709.40	129.25	40	676.70	174.26
F4	N100	40	115.10	21.26	40	113.60	20.35	40	108.03	20.82	40	102.78	19.76
	N400	40	462.73	69.02	40	453.58	82.06	40	485.15	80.93	40	467.28	111
	P100	40	67.80	14.37	40	71.25	17.65	40	70.08	16.83	40	78.25	21.89
	P200	40	171.68	24.74	40	170.33	21.06	40	174.35	27.15	40	166.03	27.66
	P300	40	248.85	35.33	40	234.08	36.83	40	244.93	47.13	40	236.53	51.66

	LNP	40	666.23	40.70	40	643.48	83.90	40	659.30	98.22	40	632.20	139.06
	LPP	40	750.00	82.52	40	735.58	120.87	40	713.18	120.50	40	655.05	153.62
Fz	N100	40	116.85	21.23	40	130.60	115.74	40	123.40	93.82	40	148.23	179.58
	N400	40	465.93	68.42	40	461.40	91.05	40	490.25	63.93	40	501.20	98.23
	P100	40	67.93	14.65	40	83.20	92.91	40	83.68	107.98	40	112.28	150.81
	P200	40	170.28	24.05	40	187.53	108.06	40	186.20	83.44	40	215.65	168.43
	P300	40	252.35	33.00	40	255.78	99.64	40	251.95	79	40	280.15	147.19
	LNP	40	681.13	46.49	40	661.58	55.81	40	669.68	59.63	40	682.48	69.51
	LPP	40	725.95	91.48	40	750.75	77.97	40	709.48	79.30	40	709.48	79.30
FC3	N100	37	117.6	21.87	37	117.14	23.11	40	114.95	21.21	40	112.83	29.39
	N400	37	468.57	72.48	37	441.27	77.48	40	472.30	68.02	40	465.83	86.63
	P100	37	69.81	14.69	37	72.35	23.22	40	71.93	20.58	40	77.05	37.1
	P200	37	173.57	25.09	37	169.24	26.49	40	177.70	21.09	40	173.10	23.52
	P300	37	245.27	33.23	37	237.05	31.27	40	236.08	35	40	234.80	39.62
	LNP	37	679.92	44.94	37	647.78	80.56	40	670.15	93.84	40	648.65	128.64
	LPP	37	711.35	90.29	37	732.08	112.61	40	694.38	110.81	40	654.45	137.12
FC4	N100	37	116.22	20.14	37	124.95	31.7	40	115.38	26.05	40	113.20	44.58

	N400	37	469.05	65.80	37	462.49	61.94	40	492.88	52.93	40	502.13	43.42
	P100	37	71.32	13.77	37	76.14	35.62	40	78.55	36.56	40	88.93	49.26
	P200	37	173.65	24.72	37	172.49	27.78	40	179.50	21.88	40	181.65	29.34
	P300	37	241.92	35.92	37	239.03	40.83	40	261.03	57.01	40	264.10	81.51
	LNP	37	675.00	41.8	37	647.49	58.47	40	677.85	59.65	40	659.28	79.31
	LPP	37	719.11	91.76	37	731.51	100.51	40	721.95	86.62	40	666.48	92.16
FCz	N100	37	118.87	20.89	37	119.49	49.03	40	121.76	58.80	40	134.27	109.58
	N400	37	465.14	72.99	37	457.32	76.11	40	493.49	68.71	40	492.95	64.73
	P100	37	71.54	13.51	37	78.03	54.19	40	81.35	64.39	40	104.46	112.21
	P200	37	169.35	24.75	37	175.70	84.07	40	187.57	69.91	40	211.51	128.13
	P300	37	247.3	34.48	37	244.6	72.94	40	260.62	86.6	40	271.62	110.39
	LNP	37	687.35	44.28	37	657.03	49.91	40	688.84	56.75	40	679.11	56.86
	LPP	37	717.32	86.89	37	740.32	75.25	40	716.05	73.22	40	674.68	70.00