T.C.
FATIH UNIVERSITY
INSTITUTE OF BIOMEDICAL ENGINEERING

INVESTIGATION OF EEG SIGNALS OF PANIC DISORDER PATIENTS DURING DIFFERENT AUDITORY STIMULI

PINAR KARAMIKOĞLU

MSc THESIS

BIOMEDICAL ENGINEERING PROGRAMME

İSTANBUL, JANUARY/ 2015 (DEFENSE)

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T.C.
FATİH ÜNİVESİTESİ
BİYOMEDİKAL MÜHENDİSLİK ENSTİTÜSÜ

FARKLI İŞİTSEL UYARANLAR SIRASINDA PANİK BOZUKLUĞU HASTALARININ EEG SİNYALLERİNİN ARAŞTIRILMASI

Pınar KARAMIKOĞLU

YÜKSEK LİSANS TEZİ
BİYOMEDİKAL MÜHENDİSLİĞİ PROGRAMI

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Pınar KARAMIKOĞLU, a **MSc student** of Fatih University **Institute of Biomedical Engineering** student ID520112022, successfully defended the **thesis** entitled **"Investigation of EEG Signals of Panic Disorder Patients During Different Auditory Stimuli "**which she prepared after fulfilling the requirements specified in the associated legislations, before the jury whose signatures are below.

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……**Prof. Dr. Sadık KARA**

Director

Date of Submission: 01 January 2015

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To my lovely family and advisor,

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January 2015 Pınar KARAMIKOĞLU

TABLE OF CONTENTS

Page

LIST OF SYMBOLS

φ	Mother wavelet
$\sum$	Sum
ψ	Positive number

ABBREVIATIONS

BAI : Beck Anxiety Inventory
CBT : Cognitive-behavioral therapy
DNA : Deoxyribonucleic acid
DSM : Mental Illness of Descriptive and Statistical Reference Book
DWT : Discrete Wavelet Transform
ECG : Electrocardiography
EEG : Electroencephalography
EMG : Electromyography
EOG : Electrooculography
GABA : β-aminobutyric acid
ICD : World Health Organization Mental and Behavioural Disorders Classification
M : Music
MAO : Monoamine oxidase
MRI : Magnetic resonance imaging
N : Noise
PD : Panic Disorder
PET : Positron Emission Tomography
PFC : Prefrontal cortex
R1 : Resting 1
R2 : Resting 2
R3 : Resting 3
SNRI : Serotonin noradrenalin reuptake inhibitors
SSRI : Serotonin reuptake inhibitors
Std. Dev: Standart Deviation
WD : Wavelet Decomposition
WHO : The World Health Organization
WT : Wavelet Transform

LIST OF FIGURES

Page

LIST OF TABLES

Page

SUMMARY

INVESTIGATION OF EEG SIGNALS OF PANIC DISORDER PATIENTS DURING DIFFERENT AUDITORY STIMULI

Pınar KARAMIKOĞLU

Biomedical Engineering Program

MSc Thesis

Advisor: Assist. Prof. Dr. Saime AKDEMİR AKAR

The aim of our study was investigating distinctive features between panic disorder patients and controls based on EEG signals with the help of advanced engineering methods. In this study, EEG data were recorded from patients with panic disorder and healthy individuals during resting, disturbing, and relaxing auditory stimulation periods. Two type of auditory stimulus were used. Recorded were decomposed into sub bands such as alpha, beta, delta and theta with using Wavelet Decomposition (WD) and Shannon Entropy (SE) and these values are calculated in each sub-band, and these values were compared with the values of healthy controls. After signal processing, Independent t-test was used to compare extracted features in patient and control groups and Paired Sample t-test was used to compare feature differences between sequential periods in each group. In addition, the proposed project with the methods and perspectives may lead to other studies related to different psychiatric disorders.

Keywords: Panic disorder, EEG, Wavelet Decomposition, Shannon Entropy, Auditory stimuli, Statistical analysis.

FATIH UNIVERSITY - INSTITUTE OF BIOMEDICAL ENGINEERING

ÖZET

FARKLI İŞİTSEL UYARANLAR SIRASINDA PANİK BOZUKLUĞU HASTALARININ EEG SİNYALLERİN ARAŞTIRILMASI

Pınar KARAMIKOĞLU

Biyomedikal Mühendisliği Programı

Yüksek Lisans Tezi

Danışman: Yrd.Doç. Dr. Saime AKDEMIR AKAR

Araştırmamızın amacı EEG sinyallerine dayanarak panik bozukluk hastaları ve kontrol grubu arasındaki ayırıcı özelllikleri, ileri mühendislik metodlar yardımıyla araştırmaktır. Bu çalışmada, EEG dataları, sağlıklı bireyler ve panik bozukluğu olan hastalardan dinlenme, rahatsız edici ve rahatlatıcı işitsel uyaranlardan oluşan periyotlar sırasında kaydedildi. İki tip işitsel uyaran kullanıldı. Kaydedilen sinyaller dalgacık dönüşümü ve shannon entropi kullanılarak alfa, beta, delta ve teta gibi alt bantlara ayrıldı ve sağlıklı kontrollerin değerleri ile karşılaştırılan bu değerler her bir alt bantta hesaplandı. Sinyal işlemeden sonra, hasta ve kontrol grupları arasında çıkarılan özellikleri karşılaştırmak için bağımsız t-testi kullanıldı ve her bir grupta ardışık periyotlar arasındaki özellikleri karşılaştırmak için eşlenik t-testi kullanıldı. Ek olarak, önerilen metodlar ve yöntemler ileride yapılacak olan psikiyatrik rahatsızlıklarla ilgili çalışmalara bir örnek teşkil etme amacı taşımaktadır.

Anahtar kelimeler: Panik bozukluk, EEG, Dalga analizi, Shannon Entropi, İşitsel uyaranlar, İstatistik Analiz.

FATİH ÜNİVERSİTESİ -BİYOMEDİKAL MÜHENDİSLİK ENSTİTÜSÜ

CHAPTER 1

INTRODUCTION

1.1 Purpose of the Thesis

To diagnose panic disorder, there are not any laboratory tests or radiological images. The aim of our study was investigating distinctive features between panic disorder patients and controls based on EEG signals with the help of advanced engineering methods. Experts mostly are grading with different scores, so it causes a problem to decide about disorder for the patient. Therefore, in this thesis it is aimed to look for electrophysiological methods to diagnose panic disorders. It will help to understand the degree and treatment of panic disorder.

1.2 Thesis Overview

In Chapter 1 some brief information about panic disorder, purpose of the thesis and what chapters include are summarized.

It is described what mental disease is and panic disorder which is one of its sub branches, some information about panic disorder such as classification and defining feature, diagnosis, treatment, etiology and epidemiology **in Chapter 2.**

Chapter 3 includes the methodology study of this study. How signals were collected from subjects, procedure of data collecting, measurement system, information about EEG, signal processing, transformation, and analysis methods were explained with details.

The results part are included **in Chapter 4.** Analysis and statistical results of the study were explained briefly. Graphs and tables were used to give detailed information. The discussion part, some recommendations were given and the thesis were concluded **In Chapter 5**.

CHAPTER 2

2.1 Psychiatric Disorders

The World Health Organization (WHO) decribes to be healthy in its own constitution, "Health is only not lack of disease and mal formation, it is well-being of body, soul and social aspects. Despite all this, mental health and mental disorders are a large part of our world as important as physical health [4]. Mental illness affects about 450 million people each year and it is a cause of four diseases of the 10 most important illnesses which are caused to disability. The rate of this continuous growing disease cause poverty, disability and economics loses. Mental and behavioral disorder are 12% limit of the global disease [5]. Epidemiological data shows that psychiatric disorders are common illness in the society. Anxiety disorders [6], depressive disorders and abuse of alcohol or other substances are common psychiatric disorders in the developed western countries [5].

Today, many psychiatric disorders are the fact that it is a brain disease. Very large rate of population are neglected or ignored. As a result of this position, the most of disease has missing treatment because of increasing of mental disorder. Small part of this population can reach the true therapy [4].

DSM (Mental Illness of Descriptive and Statistical Reference Book) and ICD (World Health Organization Mental and Behavioural Disorders Classification) are used for diagnosis in psychiatry. According these, mental illness are categorized and clustered [7].

2.1.1 Anxiety Disorder

The mean of anxiety is derived from anger is meaning that “blockage”,”get tight”and “drowning”. Another conception is fear that is mentioned with anxiety. Fear is a term from German. The word of origion in this language means waiting, to lie in ambush and attack [8].

Anxiety is the sense which is similar to the fear and people have this feeling occasionally. The person perceive as having bad news, living disaster lack of reason, a non-specific distress and feeling anxiety [9, 10].

Anxiety is sophisticated and widespread disorder. It is related with crucially morbidity and social cost [11]. Anxiety disorders are more current in women than they are in men [12] and the lifetime prevalence of anxiety is 13.6% in Europe [13]. Actually anxiety is pathological condition that partners' somatic symptoms are based on hyperactivity of the autonomic nervous system. Actually anxiety is pathological condition that partner's somatic symptoms are based on hyperactivity of the autonomic nervous system. It separates from fear in response to the cause [14]. Anxiety is limited expectation of danger unlike scare [15].

2.1.2 Symptoms of anxiety

It is under two headings are physical and psychological in symptomatology. Tremors, chills, back and headaches, muscle tension, hyperventilation, fatigue, startle response, flushing and fading, tachycardia, palpitations, sweating, cool hands; diarrhea, dry mouth dryness, frequent urination, paresthesia and difficulty in swallowing are physical symptoms. Psychological symptoms include a feeling of fear, difficulty in concentration, hypervigilance, insomnia, decreased libido, knotted feeling in the throat and stomach are feeling contractions. Psychological symptoms include a feeling of fear, difficulty in concentration, hypervigilance, insomnia,decreased libido, knotted feeling in the throat and stomach are feeling contractions [14].

Anxiety is most comman disoder in population. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) restructured the anxiety disorders into these groups:

Anxiety disorder;

- Panic disorder
- Generalized anxiety disorder
- Social anxiety disorder
- Agoraphobia
- Specific phobia

- Separation anxiety disorder
- Selective mutism
- Substance/medication induced anxiety disorder
- Anxiety disorder due to another medical condition [16].

Panic disorder is described is common disease as a person consult to healty care is in between first step. Panic disorder is distinguished as new category from anxiety neurosis by Klein. He distinguishes panic attacks which respond to imipramine is tricyclic antidepressant and generalized anxiety which not respond to imipramine. Based on this distinction, panic disorder is categorized. This is pharmacological dissection [17].

2.2 Panic Disorder

Panic (Panikos) word is derived from Pan of Greek God. In encyclopedia, it defined as horror, fright without rational reason in person or a community. The upper body of Pan is human; the lower past is god of herds of goats. It's fear appearance cause the escape of nymphs of water and forest [18].

Anxiety disorders, the most current psychiatric conditions. This disorder is huge part of scientific researchs. All the same, panic disorder is the best-studied anxiety disorder. It has high rate of lifetime prevalence [19, 20]. Panic disorder is based on either biological or psychological disease [21]. This is related with increased risk under the anxiety disorder [22].

Patients with panic disorder develop an icrease awareness and fear againts some variations unlikee another anxiety disorders. In patients with panic disorder, sensitivity of anxiety loses for late time or it never loses. The lack of get used to worry is said in patient. Normal person as physiological get used the stimulus of anxiety, but panic disorder patient is not [23]. It is a common psychiatric illness that causes noteable short- and long-term morbidity [24]. This disorder is associated with important escalation in life quality and psychosocial conditions [25]. Panic disorder is usually onset between pubescence and 30 years. The avarage of onset age is 25 [26]. Panic disorder is very effective illness. It affects up to 5 % of the population [19]. Panic disorder is related with of a high percentage of social, physical and vocational disability secondly being health complaints [27].

On the other hand, panic disorder is uncomfortable situtation of anxiety life. It can be acute or intense experiences. It is acceptable appearance psychological of panic and its attack related with behavior about escape. The reason of escape is to release panic attack. Patients can escape. At the same time panic disorder patients can not escape. Instead of escape, they can walk or talk at the al time. This anxiety is usually anticipatory type. It reason general arousal state of continuity in patient with panic disorder [8].

Panic attack is criteria for panic disorder. PD consists of unexpected attacks [28]. It must occur more than 1 month of subsequent worry about another attacks, or behavioral changes related to the attack according to the Diognosis and Statistical Manual of Mental Disorder, Fourth Edition [29]. These attacks have anxiety, fear, autonomic and respiratory symptoms. When the patient have panic attacks, they show respiration problem [28]. Actually, The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition *(*DSM-5*),* have no important changes. Panic attacks change into 2 categories which are expected and unexpected attacks [16].

Physiological things are symptoms of general anxiety. These attacks may takes from ten minutes to several hours. Time and place are not determined. After attack, patients sense profound fatigue and weakness. On the other hand some patients may sleep for hours. Nocturnal panic atttacks awaken the person from sleep [30].

Panic disorder is heterogen disease which has panic attacks class noctural, respiratory, vestibular, cognitive and fearless according to different symptom clusters [31]. Noctural Panic attacks which increases the severity of the disease [32]. The respiratory subtype of panic atttacks is shortness of breath, chest pain, fear of death, choking sensation, numbness [33]. Panic attacks that to put patient out about professional and social functioning occur for 15-20 minutes. It sometimes goes on approximate a hour [34].

2.2.1 Etiology

The reason of panic disorder is not defined. This is accepted the product of psychological, behavioral, and biological forces. It is combined stress and conflict. This disorder is genetically influenced.

It is related with biological condition strongly. Figure 2.1 shows that brain structure change in patients with panic disorder [35, 36]. The amygdala is known to have 13

nuclei. It is categorized into 3 groups which are lateral, basal and central subregions [37]. A nucleus of the amygdala is separeted into groups are laterobasal subgroup, centromedial subgroup, and cortical subgroup. These are related with fear [38]. The lateral subgroup receives information from the cortical and subcortical areas, the basal subgroup inter-connects the lateral and central subgroups, and sends the output to the cortical areas, and the central subgroup conveys the information to the brain regions including hypothalamus and periaqueductal gray [38]. The laterobasal and central subgroups are also connected with bed nucleus of the stria terminalis, which also projects to hypothalamus, cerebellum, and brain stem areas [3].

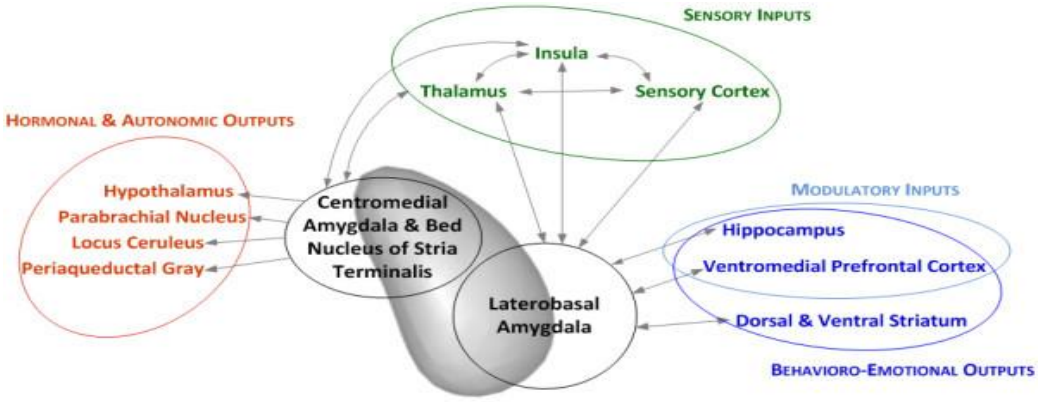


Figure 2.1 Schematic diagram of inputs to outputs from the amygdala, relevant to panic disorder pathogenesis [2, 3]

The relation is occurred in the locus caeruleus, β-aminobutyric acid (GABA) system benzodiazepine receptorcomplex, septohippocampal region, and ventromedullary center [39]. In central nervous system, 50 % of noradrenergic receptors are the locus caeruleus. When this area is stimulated, sympathetic arousal and an outpouring of catecholamine are generated. It is one of panic disorder symptoms [40]. GABA is a neurotransmitter. This neurotransmitter reduces excitability of neuron. GABA has a result hyperpolazition of neuron. It reduces in anxiety. Benzodiazepines are effective for GABA. They improve the action of GABA. So it produces a calming influence [41]. The septohippocampal region supply to moderate input from external and internal environments. Mismatch of a input cause activity inhibition [42].

Septohippocampal region may be hypersensitivity in panic disorder patients. The mismatch between the right and left parahippocampal regions is found for panic

disorder patients according to the tomography studies. On the other hand, it isn't found in patients lack of panic disorder [43].

In patient with panic disorder, positron emission tomography represent of increasing of flow of the right parahippocampal region and reducing of serotonin type 1A receptor binding in the anterior and posterior cingulate [44]. Magnetic resonance imaging (MRI) evidence panic disorder patients have smaller temporal lobe volüme despite normal hippocampal volume [45].

Some studies show that, chronic hyperventilation and carbon dioxide receptor hypersensitivity cause panic disorder [46]. A significant blood-barrier that is responsible for PCO2, pH, and acetate levels is not found in the ventromedullary center in the blood. The sensivity of carbon dioxide and lactate levels is very great in panic disorder patients than the person lack of panic disorder [47].Panic disorder is the anxiety disorder. It is best-showed passage of genetic according to studies having a panic disorder.

In first and second kinds of panic disorder patients defined 4 or 10 times [48]. It is genetically inherited neurochemical dysfunction. The genetic rates are estimated to 0.3 % to 0.6 %. According to the segregation analyses, there is no distinct deoxyribonucleic acid (DNA) linkages are known. All the same time, some chromosomal regions like 13q, 14q, 22q, 4q31- q34 and probably 9q31 are related with phenotype of panic disorder according to the genetic studies [49]. On the other hand, culturel and unfavorable environment may be effect having a panic disorder in addition to genetic factors [48].

2.2.2 Natural History

Panic disorder has varies stage. These are six stages. Chronicity is generally found in this disorder [50].

Some panic disorder patients may stay in one stage, whereas other patients may progress rapidly through all six stages. Half of the patients stay in stage 1 that is limited-symptom attack. These patients show less than the four signs needful for diagnosis of panic disorder. The other half of panic disorder patients are in stage 2. This stage has panic attacks. In stage 2, patients have four or more of the symptoms according to

researchs, the patients are in stage 1 and 2, properly treated and the panic disorder is less likely to progress [51].

The third stage of this disorder is hypochondriasis. A patient in this stage, concerns about medical illness, despite medical assurances. The panic attacks may depend on with environmental stimuli. It is determined as phobic avoidance behavior. This is known as phobic avoidance behavior. Driving car, going to stores or shopping malls are the most frequent fears 19. The phobic avoidance like agoraphobia improve in stage 4 [52]. The other stage is 5. Some phobic behaviour develops in this stage. The patients are in this stage, may become housebound. The final stage is 6 that is secondary depression. It is believed to result from progressive disability a demoralization [53].

2.2.3 Classification and Defining Features

Panic disorder is characterized by recursive, unexpected panic attacks. Three or fewer symptoms determine limited-symptoms attacks whearas four or more of the symptoms describe the full-blown attacks in Table 2.1. The frequency and intensity of panic attacks vary among patients. Table 2.2 defines the three types of panic attacks found in patients which have panic disorder [29].

Panic attacks can be seen in many cases disease like panic disorder [54]. But for diagnosis of panic disorder, panic attcaks progress spontaneously and it must be repetitive. These attacks look like showing escape or war reaction of patients. But really and trigger stimulation is absent for panic attack [55].

Table 2.1 Symptoms of panic attack

Symptoms of panic attack;

A panic attacks ocur spontenously. They reach a peak within 10 minutes lack of hazard. The four or more of the following symptoms ocur during attack:

- palpitations, pounding heart or accelerated heart rate
- sweating
- trembling or shaking
- sensation of shortness of breath or smothering
- feeling of choking
- chest pain or discomfort
- nausea or abdominal distress
- feeling dizzy, unsteady, light-headed or faint
- derealization(feeling of unreality)or depersonalization(feeling of being detached from oneself)
- fear of losing control or going crazy
- fear of dying
- chills or hot flushes

Table 2.2 Major three types of panic attacks

Situaitionally bound (cued) Panic	Almost always occurs nearlyupon encountering,	A patient who always panics when in a crowded shopping mall	Frequent in panic disorder.Experienced by the majority of patients with social and specific phobias
Situationally predisposed panic	Often, but not always, occurs in response to a situational cue	A patient who is more likely to panic when standing in a supermarket line	Frequent in panic disorder. Experienced by many patients with generalized anxiety disorder and post-traumatic stress disorder
Unexpected panic	Appears (to the patient) to occur spontaneously or ‘out of the blue’	A patient who panics but can’t identify any trigger for the attack	Necessary for diagnosis of panic disorder

Panic disorder generally co-ccurs with agoraphobia [56]. Panic disorder with or without agoraphobia tell the concept of an acquired fear of bodily sensations, particularly sensation related with autonomic arousal [57]. It associated with significant escalation in quality of life [58]. Table 2.3 defines the DSM-IV diagnostic criteria for panic disorder. The panic attacks must not based on only effects of psychoactive substance (intoxication or withdrawal), medication or a general medical condition (e.g. hyperthyroidism, vestibular dysfunction).

Panic disorder with agoraphobia (PDA) is a very psychosocial problem for individuals and society [59]. Panic disorder lack of agoraphobia (PD) is characterized by recurrent panic attacks and panic disorder with agoraphobia (PDA) includes besides these attacks avoidance of their triggering situations. Panic disorder with agoraphobia may start with the panic attacks.

Table 2.3 Diagnostic criteria for panic disorder and agorafobia [29]

Diagnostic criteria for panic disorder and agorafobia

Panic disorder;

- One or more full-blown panic attacks, occurring in the absence of real hazard
- The attacks are not due to a general medical condition
- Attacks are followed by a month or more of any of the following:
    - insistent worry about having more attacks
    - worry about the influences or consequences of the attacks
    - behavioural changes as a result of the attacks (e.g. avoidance of work or school activities)

Agoraphobia;

- Anxiety about being in places or situations from which escape might be difficult or embarrassing
- Avoidance of a wide range of situations, including:
    - being outside the home
    - being alone at home
    - bridges
    - elevators
    - travelling by car, train, bus or aeroplane

2.2.4 Epidemiology

The prevelance rate of panic disorder is very high in community [60]. A trouble factors are in the course of panic disorder prevalance rate of 3.5 % [61]. PD without agoraphobia has 3.7% prevalence rate, and PD with agoraphobia has 1.1% prevalence rate. The prevalence of panic disorder is high. Despite this prevalence, it is relatively low in community. It is nearly 2.7% for 12 month. The prevalence rate of primary care in society substantial higher. It is raughly 6.8% [62, 63].

Panic disorder is effective raughly 1–5% of adolescents [20]. Panic disorder (PD) is a prevalent anxiety disorder affecting 1–8% of the U.S [64]. Panic attack's prevalance rate is 3.5-6 %.

Panic disorder has generally with mood symptoms and mood disorder. For example prevalence rates of major depression may be as much as 50-60% lifetime. Panic disorder may share important comorbidity like chronic obstructive pulmonary disorder, irritable bowel syndrome, migraineheadache, obsessive-compulsivedisorder, restlessleg syndrome, fatigue, specific phobias, social phobia and agoraphobia [65, 66].

Cardiovascular disorders like mitral valve prolapse, hypertension, stroke are also comorbidities with panic disorder [67]. Panic disorder patient with coronary disease, they may have myocardial ischemia during panic event [68, 69].

Panic disorder bring back sudden death [70]. However, 30 % panic disorder patients have chest pain and normal finding on angiograhy. 5-40 % of patient with asthma have panic disorder. 15% of patients with headache, 20% of patients with epilepsy, and 10% of patients in primary care settings have also panic disorder. Additionaly, panic disorder patients have low oxygen consumption and low exercise tolerance together with general population [71].

The rate of substance abuse with panic disorder in patient is 7-28 %. The risk is 4-14 times greater than normal population. The 8-15% people who are in alcohol treatment program have panic disorder. Pregnant woman may have panic disorder like preterm labor and infants of smaller birth-weight for gestational age [72]. Women effect from panic disorder than men. The prevalence estimates is 0.7 % for women, on the oher hand it is 0.3% for men at one-month time. Panic is more widespread than men and

women who are pregnant or during the postpartum period have lower panic than normal situtation. But it is less current during pregnancy. Panic disorder can occur any age in people. Especially it generally develops between 18- 45 ages. If the onset age of panic disorder is late, people have lower comorbidity, hypochondriasis. They have better handle than panic disorder occurs in early age [73].

2.2.5 Treatment

Panic disorder is very serious problem and it undermines the quality of life of individuals. In recent years, there are wide studies about pathophysiology and treatment of panic disorder. In result of these studies, effective method occur for panic disorder [74]. Before 1980s, treatment wasn't usually effective. Three-quarters of patients could not get well [75].

General anxiety disorder and panic disorder require long-time treatment. Although after a long-time treatment, symptoms of disorder may go on persistently [34]. Patient education, behavioral therapy and pharmacologic therapy are avaliable for treatment of panic disorder. Psychotherapy effect is not indicated for treatment of panic disorder [76].

Before starting treatment; doctor should listen, supply a common terminology between patient-doctor. Details of disease is given to patient and doctor should give information of side-effects, if the pharmacological treatment is applied to patient [77]. The psychopharmacological treatment has some phase according to some studies up to date and experiences of clinicians. These are;

1. Acute phase of treatment,

2. Stabilization phase,

3. Maintenance phase,

. Acute phase of treatment: This phase includes elimination of panic attacks, decreasing anxiety and phobic complaining significantly and furthering the highest good condition to patient.

2. Stabilization phase: This phase takes 2-6 months. The main goal is to prevent patients from environment can be created attacks and behaviour of avoidance. In a result, to reach the patient healty life is aimed.

3. Maintenance phase: When the patient responds to acute treatment, sustaining the treatment for a few months is ectremely important. This phase supply to toughen benefits which gained in acute phase, isolation of social life and get over to behaviour of avoidance [34].

Cognitive-behavioral therapy (CBT) or one of four types of medications are approved in the American Psychiatric Association's treatment guidelies for treatment of panic disorder [78]. Years have shown that CBT is an effective treatment for panic disorder [79] that is with agoraphobia or without agoraphobia [80]. It includes interoceptive exposure is the psychological treatment of choice for panic disorder [27]. This treatment is most widely used for mental disorder like panic disorder according to more than 50 years [81]. However sometimes CBT may not be effective because of some reasons which are long distances for weekly appointment in rural areas. This extra travel time may create some problems like financial and rejection to the treatment [82]. At the same time the general and specific neurobiological effects of CBT are still widely unknown [83].

Additionally pharmacotherapy may also be beneficial for panic disorder despite not producing significant utility of the combination of cognitive-behavioral treatment with medication, either treatment alone [84]. That is to say cognitive–behavioral psychotherapy and pharmacological options are therapeutical strategies. Serotonin reuptake inhibitors (SSRI), serotonin noradrenalin reuptake inhibitors (SNRI), tricyclic and benzodiazepines [85].

Drug treatment is effective for panic disorder patients in the short-term. It is nearly 6-8 weeks. Benzodiazepines and antidepressants have been established for treatment. Effect of long-term drug treatment is not adressed. Most data is based on benzodiazepines for long-term efficieny. Effects of triazolobenzodiazepine, alprazolam are reviewed together with some other effective drugs like clonazepam. On the other hand tricylic antidepressants are also effective for a long-time treatment of panic disorder. Monoamine oxidase inhibitors is not clearly determined[86]. On the other hand, some drugs are used with together, that time the efficiency can occur like using tricyclic antidepressants and monoamine oxidase inhibitors which have blocking panic attacks for treatment of panic disorder and agoraphobia [87]. Actually lately, drug treatment which is based on benzodiazepines is not effective for treatment of panic disorder with/withoutagoraphobia. As noted tricyclic antidepressantsand monoamineoxidase

(MAO) inhibitor antidepressants are effective beside benzodiazepines which are best-known [88]. Benzodiazepines were used for limited efficiency until using alprazolam that is most widely used benzodiazepines. It is approved for panic disorder in United States. Clonazepam is high-potency benzodiazepines like alprazolam. At the same time it is used for generalised anxiety disorder. However it is effective during sedation daytime [89]. Lorazepam and diazepam are less potent agents. They may be effective using with higher doses to be antipanic agents [90]. When the benzodiazepines are used for long-term treatment, their doses should be increased to treat panic and agoraphobic disorder. If benzodiazepines are used for short term treatment, they are useful for anxiolytic or hypnotic purposes. Benzodiazepines also produce a variety of side effects. They are sedation, reduced coordination, and impaired cognition which change according to quantity of dose and duration of treatment [91].

2.3 Panic Disorder and Electrophysiology

Psychiatric conditions derived from psychological processes [92]. Anxiety disorders are related with an increase in cardiovascular mortality. Researches prove increased heart rate (HR) and QT interval variability and a relatively increased sympathetic function in anxiety [93]. Anxiety disorders like panic disorder have abnormalities autonomic control [94]. Panic disorder patients have panic attacks. These attacks are related with several autonomic symptoms, chest pain, heartpounding, tachycardia, and shortness of breath. These conditions are associated with autonomic dysfunction. Recent studies show that this relationship increase risk and anxiety cause death and cardiovascular mortaliy [95]. EEG signals reflect the collective activity of brain cells. Generally EEG signal occıır with four main waves which are alpha waves (8-13 Hz), beta waves (13-30 Hz), delta waves (0-4Hz) and theta waves (4-7 waves). These waves in EEG, show any pathological condition about brain [96]. Since the 1980s, a high rate EEG abnormality has been reported for patients with PD. It is approximately 15-30% [97, 98]. Brainstem evoked potentials (BEP) and event-related late evoked potentials are studied in panic disorder with EEG. So far the studies show that brainstem and limbic region are related with occuring panic disorder [99]. Topographic EEG abnormalities are widespread in patients with panic disorder and they don't focus on single part. These neuroelectric finding are related with brainstem in subcortical region [100]. However EEG studies in panic disorder show brain abnormalities but exactly structure of brain can not be stated

[101]. 29% non-epileptic EEG abnormalities are found in other study, and these abnormalities of limbic system are proven in MR [101]. Abnormalities of EEG are related with temporal lobe show the parahippocampal asymmetry in PET [102]. Lepola and his friend prove that panic disorder patients have 24% non-epileptic EEG abnormalities [103]. EEG abnormalities can be found in panic disorder patients. Stein and Uhde states 14% EEG abnormalities which are not epileptic in patient with panic disorder [97]. Beauclair and Fontaine determine EEG abnormalities that are epileptic ¼ of patients with panic disorder [104].

CHAPTER 3

MATERIAL &METHOD

In this section, information about subjects, the procedure of research such as

Process of experiment, auditory stimuli, EEG registration and signals processing methods are included.

3.1 Subjects

10 patients were taken from Bezmialem Vakıf University Faculty of Medicine Department of Psychiatry and controls were taken from people of Fatih University. The protocal was approved both university and Bezmialem Vakıf University Faculty of Medicine. Made for this study was Bezmialem Foundation ethical approval from the university. (Appendix A). 10 patients who are diagnosed with panic disorder according to DSM-IV criteria and the patients were diagnosed and evaluated by Dr.Erdem Deveci. Ten age-matched control subjects were involved for this study as shown table 3.1. Volunteers signed concent form to participate this experiment. (Appendix B). Volunteers filled the questionnaire of sociodemographic characteristics (Appendix C), Beck Anxiety Scale Test (Appendix D) and Beck Depression Scale Test (Appendix E).

Table 3.1 Demographics and self-report measures of subjects

Features	**Panic Disorder Patients**	**Controls**
Number	10	10
Male/Female	4/6	6/4
Age (Mean±SD)	33±14,61	30.1±6.20

Inclusion Criteria:

- Between 18 – 55 years old
- Diagnosed with PB according to DSM-IV
- Giving written, informed consent

Exclusion Criteria:

- Having any other mental disorders
- Having pathological, endocrinological, cardiovascular disorders
- Psychotropic medication usage
- Cardiovascular medication usage
- Having head trauma or other neurological disorder
- Having cardiac pacemaker or any device that affect cardiac autonomic function
- Having substance abuse disorders (including alcohol abuse)
- Having hearing loss
- Being pregnant or lactation period in females
- Epilepsy history about patient or among relatives

3.2 Procedure and Auditory Stimuli

The EEG signals were recorded between 9.00 AM and 17.00 PM. In order to get the good quality data quiet and illuminated room is provided at Bezmialem Hospital, Department of Psychiatry. The subjects were quietly sitting on chair in relaxed position and they close their eyes during process like Figure 3.1. They did not move until end of the EEG recording. So some artifacts prevent during recording. BrainAmp recording program is used for EEG recording in panic disorder patients. Electrodes are attached on frontal (F3, F4), central (C3, C4), parietal (P3, P4) regions of brain. Figure 3.2 shows regions of electrodes. Each subject was exposed to 10 minutes EEG registration. Figure 3.2 shows the channels location. There are five channels that consist of 2 minutes periods. Figure 3.3 shows process of experiment. First period was resting period with 2 minutes duration and labeled with R1. Second period is first auditory stimuli period and lasted 2 minutes that are chosen as voice of an ambulance in our study [105]. It is labelled with N. Third period is resting period with 2 minutes duration and labeled with R2. Fourth period is music period that is chosen with natura sound [106]. Last one is resting period. It takes 2 minutes and it is labeled with R3. Recorded data were

decomposed into sub bands such as alpha, beta, delta and theta with using WD and SE and these values are calculated in each sub-band, and these values were compared with the values of healthy controls.

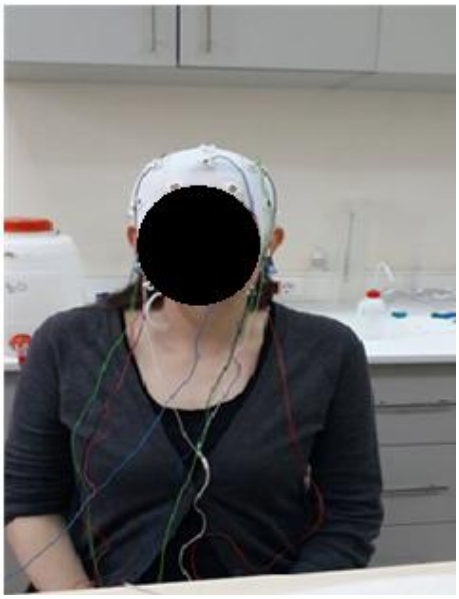


Figure 3.1 Eyes of subjects are closed during process application

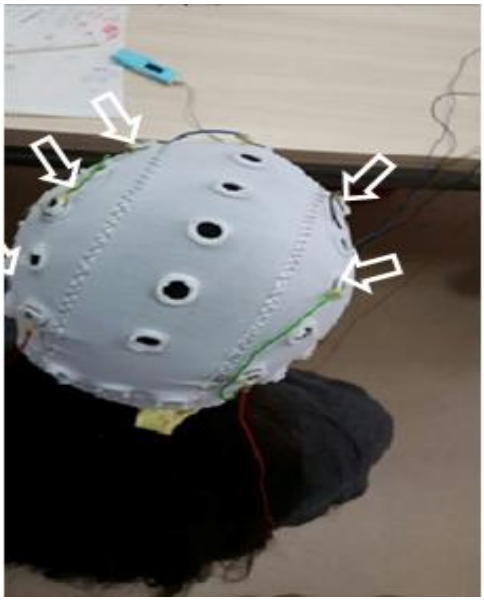


Figure 3.2 Location of channels

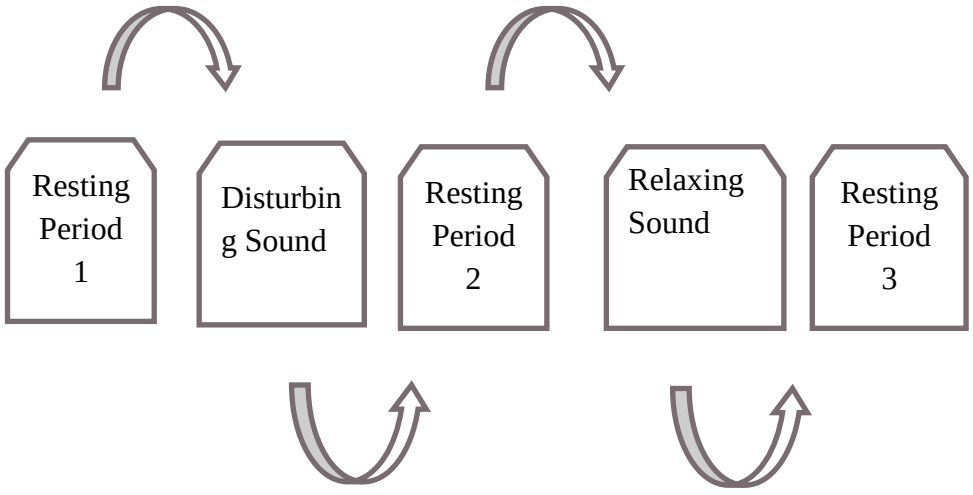


Figure 3.3 Process of experiment

3.3 Signal Description and Measurement System

V-Amp DC model of Brain Vision Product is used electroencephalogram measurements in Figure 3.4. It is used also measurement of EOG, ECG, EMG. The auxiliary ports consist of sensors for peripheral signals like GSR, blood flow, temperature interface.

Figure 3.4 V-Amp DC model of Brain Vision Product [1]

3.4 Electroencephalograph (EEG)

Hans Berger recorded electroencephalography (EEG) in 1929 for the first time. It is bioelectrical activity of cerebral by electrodes are placed on scalp [107]. A number of sensors are attached to the head and hooked by wires to a computer. The computer records the electrical activity of the brain for a long period of time [108]. EEG measures the changes of the electrical activity in term of voltage fluctuations of the brain [109]. It is also a graphic record of the activity of a huge number of neuronal-membrane potentials. It is mostly used for the diagnosis, monitoring, and management of

neurological disorders including epileptic seizures, which are characterized by frequently occurring of spikes in EEG signal [110]. However, EEG is very weak signal [111]. It is easily influenced by biologic, technological extrinsic artifacts [111] and other noise. This may be a problem for diagnosis [112, 113]. Figure 3.5 shows that EEG is the most commonly recorded according to the international 10-20 electrode placement system. The 10-20 system was developed to standardize the collection of EEG[114].

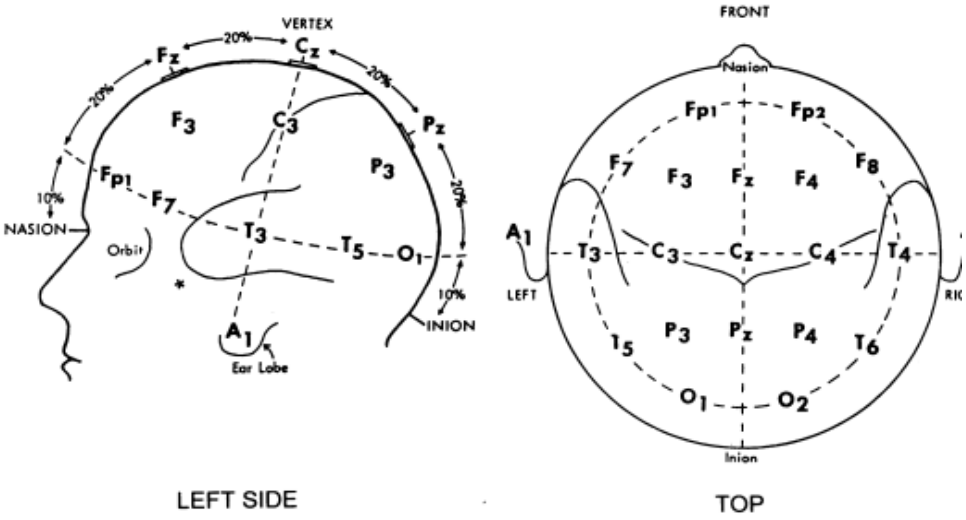


Figure 3.5 The International 10-20 electrode placement system [124].

3.5 Signal Processing

Signal processing is used for analysing the EEG data. Before signal processing, recorded data is shown Figure 3.6.

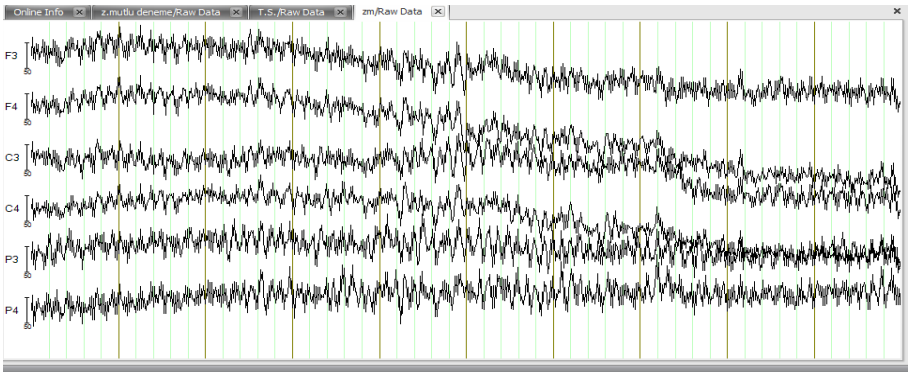


Figure 3.6 The raw EEG signal which is recorded from healthy person

3.5.1 Wavelet Decomposition

Approximation and detail components are defined by wavelet decomposition [115]. This decomposition process is renewed and it uses successive approximations. These approximations give signal that is broken down into many lower resolution components. Wavelet transform (WT) is a good preference for the area which is related with non-stationary signals [116]. Wavelet is a multi-resolution analysis which provides a good localization properties in time and frequency domain [117]. Theta, alpha, beta and gamma waves in brain can be extracted by this transformation [118]. A wavelet transform decomposes the wavelet components. These are time-domain signals and they supply more detailed information [119].

3.5.1.1 Discrete Wavelet Transform

A time-scale representation of a digital signal is occured by digital filtering techniques in a discrete wavelet transform (DWT). Different cut-off frequencies fiters are used for analyzing a signal that is on different scale [120]. The x[n] is the original signal to be decomposed and h[n] and g[n] are low pass and high pass filter and ↓ 2 denotes subsampling [121] in Figure 3.7. The f is showed at each level as the bandwidth of the signal [122]. The basic principle of wavelet theory is expressed in Gabor's paper in 194 [123].

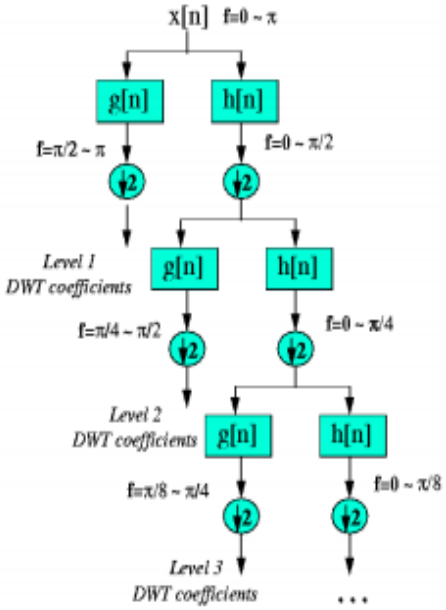


Figure 3.7 The subband coding algorithm [124]

In discrete wavelet analysis, a multi-resolution description is used to decompose a given signal $x(t)$ into increasingly finer detail based on two sets of basis functions, the wavelets and the scaling functions, as follows:

$$x(t) = \sum 2^{j_0/2} a_{j_0}(k)\varphi(2^{j_0}t - k) + \sum_{j-j_0}^{\infty}\sum_{k} 2^{j/2} d_j(k)\psi(2^j t - k)\, j_0 \qquad (3.1)$$

where functions $\varphi(t)$ and $\psi(t)$ are the basic scaling and mother wavelet, respectively. In the above expansion, the first summation represents an approximation of $x(t)$ based on the scale index of j_0 while the second term adds more detail using larger j (finer scales). The coefficients in this wavelet expansion are called the discrete wavelet transform (DWT) of the signal $x(t)$ [125].

3.6 Shannon Entropy

Entropy is defined as a measure for information theory by Claude E.Shannon. Applied to EEG analysis, Shannon entropy measures the predictability of future amplitude values of the EEG based on the probability distribution of amplitude values already observed in the signal.

It quantifies the probability density function of the distribution of values. Probability density functions are simple histograms of the amplitude values versus the number of samples at each value in the sampled signal [124]. Figure 3.8 is example of Shannon entropy. It shows that the probability density function of amplitude values is quantified. During the awake state, amplitude values vary to a greater degree compared to anaesthesia, when there are less different amplitude values.

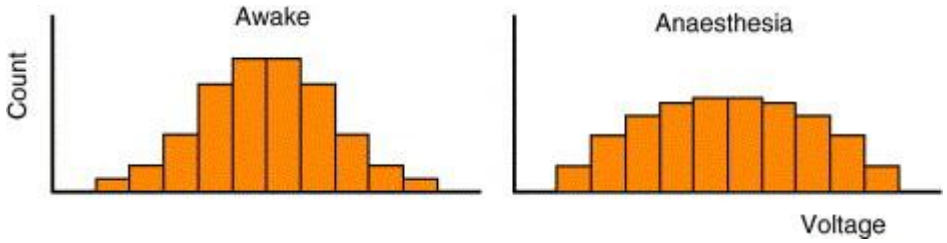


Figure 3.8 The probability density function of amplitude values during awakestate and anasthesia [126]

Information theory dealt with the nascent science of data communications. Shannon entropy (H) is given by the following equation:

$$H = -\sum p_k \log p_k \qquad (3.2)$$

where pk are the probabilities of a datum being in bin k. It is a measure the spread of the data. Data with a broad, flat probability distribution will have high entropy. Data with a narrow, peaked, distribution will have low entropy. As applied to EEG, entropy is the statistical descriptor of the variability within the EEG signal [127].

3.7 Statistical Analysis

3.7.1 The Indepent Sample Student's t-test

When two groups compared independently, independent sample student's t-test is used. In this this study, SE valuesof patients and controls groups are compared in each periods (R1, N, R2, M, R3) at F3, F4, C3, C4, P3, P4 regions by independent t-test. The test first pooled standart deviation has to be calculated by,

$$S_p^{\,2} = \frac{(n_1 - 1)s_1^{\,2} + (n_2 - 1)s_2^{\,2}}{n_1 + n_2 - 2} \tag{3.3}$$

Where $n_1 + n_2 - 2$ is the degree of freedom. The equation for calculation t value become [128].

$$t = \frac{\overline{x_1} - \overline{x}_2}{\frac{sP^2}{n_1} + \frac{sp^2}{n_2}} \tag{3.4}$$

3.7.2 Paired Sample Student's t-test

The paired sample t-test is used for samples that are in same group. The SE values of patients are compared between sequential periods by Paired sample t-test and the SE values of controls are compared between sequential periods by Paired sample t-test and produce a t value;

$$t = \frac{\frac{\sum \mathrm{d}}{N}}{\sqrt{\frac{\sum d^2 - \frac{\left(\sum d\right)^2}{N}}{N\left(N-1\right)}}} \tag{3.5}$$

In formula, d is the difference between matched samples and N is number of samples [128].

CHAPTER 4

RESULT

In this chapter, results of analyzed EEG signals exist. EEG was recorded from F3, F4, C3, C4, P3 and P4 regions of brain. These signals were recorded from 10 panic disorder patients and 10 healthy people. Recorded EEG data were decomposed into alpha, beta, theta and beta sub bands by DWT. After that, SE was calculated in each sub bands. MATLAB® software algorithms (v. 7.6.0. R2008a) was used to signal processing and SPSS® (v.20) software was used to do statistical analysis. It includes independent sample t-test which used to compare SE values in patients and healty people. At the same time paired sample t-test was used to compare values between periods in each group. We chose these tests because of feasibility of normal distribution. Before these analyses, EEG signals were recorded by Brain Amp System and Brain-Amp Software.

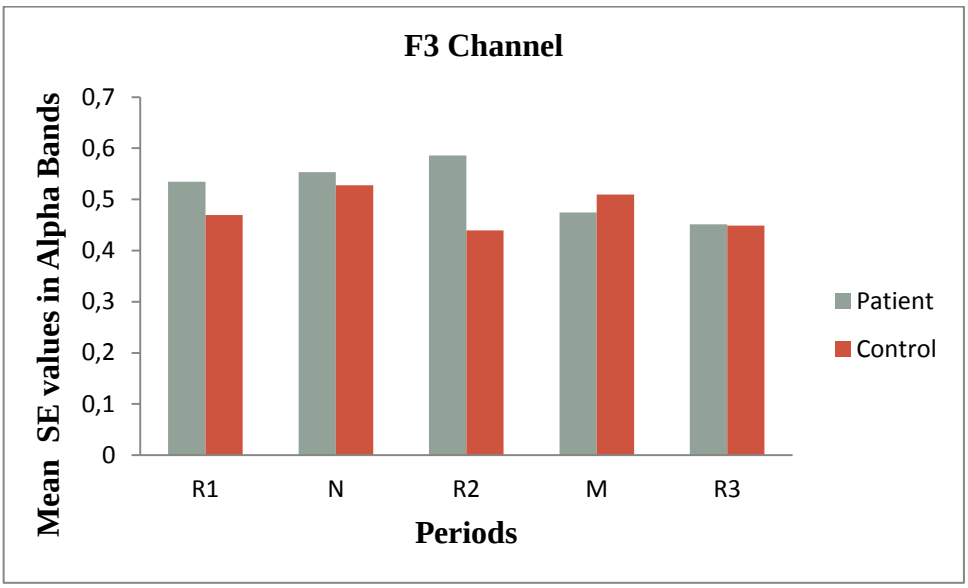
Table 4.1, 4.2, 4.3, and 4.4 show calculated SE and p values in each periods (R1, N, R2, M, R3) between patients and controls in each sub bands respectively alpha, beta, delta, theta in F3 region.

Table 4.1 Comparison of SE and p values of alpha band during all measurement periods between patients and controls in F3 channel

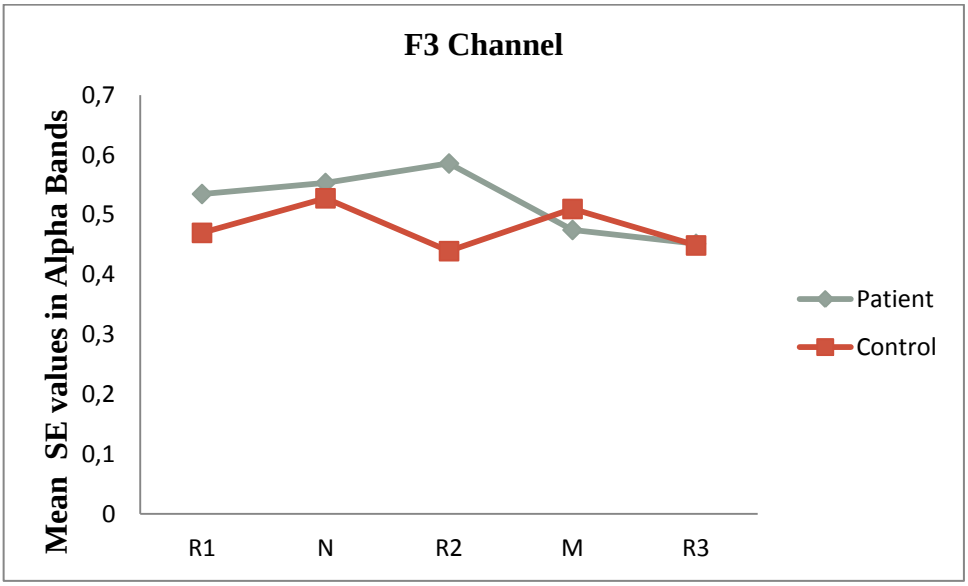
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,469710 ±0,038164	-0,534720 ±0,170145	0,254
Alpha	N	-0,527530±0,175948	-0,553136±0,196653	0,758
Alpha	R2	-0,439210±0,024073	-0,585670±0,204107	0,050*
Alpha	M	-0,509650±0,166622	-0,474570±0,162167	0,639
Alpha	R3	-0,448710±0,046617	-0,451450±0,111367	0,944

*$p \leq 0,05$ is accepted for significant difference

Table 4.1 shows that the significant difference is at R2 state for alpha band in F3 channel.



(a)



(b)

Figure 4.1 (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of F3 channel in patient and control groups

Figure 4.1 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at R1 period. While groups are listening the noise, there is no significant difference between patiens and controls but the mean of SE of patients is higher than controls at R2 periods. In M period, mean of SE of controls is higher than patients.

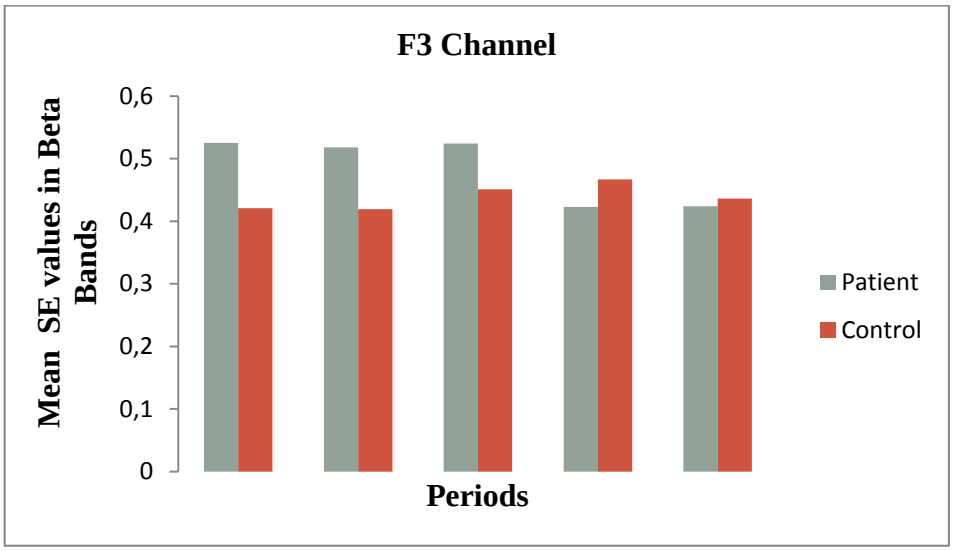
On the other hand, there is no significant difference between patients and controls in R3 periods. Values of patients and controls are equal at R3 state.

Table 4.2 Comparison of SE and p values of beta band during all measurement periods between patients and controls in F3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,238490±0,093198	-0,429070±0,246209	0,042*
Beta	N	-0,230850±0,068005	-0,435370±0,291755	0,056*
Beta	R2	-0,238540±0,140543	-0,462890±0,301840	0,053*
Beta	M	-0,239990±0,078559	-0,379890±0,208612	0,063*
Beta	R3	-0,219750±0,051100	-0,386270±0,219608	0,031*

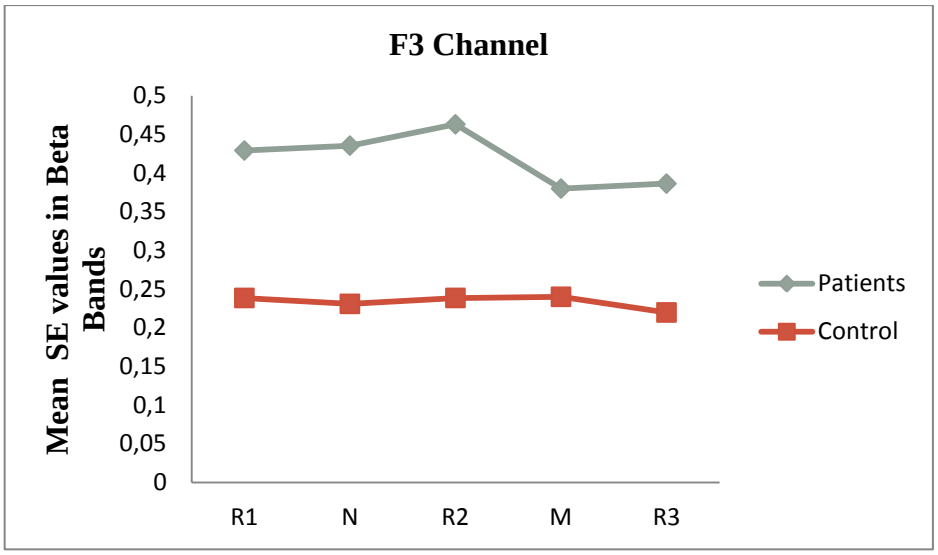
*p≤ 0, 05 is accepted for significant difference. p ≤ 0, 08 is accepted closest value for significant difference

Table 4.2 shows that the significant difference is at all state for beta band in F3 channel.



(a)

Figure 4.2 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of F3 channel in patient and control groups



(b)

Figure 4.2 (continue) (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of F3 channel in patient and control groups

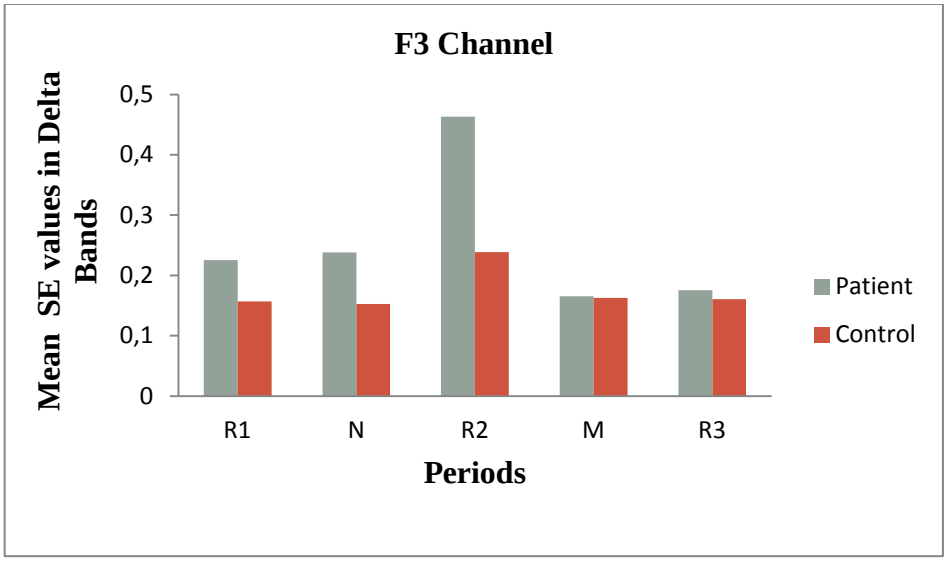
Figure 4.2 shows that red column and line graphs indicate controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at all periods. The mean of SE of patients is decreasing after R2 state. While the groups are listening the relaxing music, only mean of SE of patients decrease at M state.

Table 4.3 Comparison of SE and p values of delta band during all measurement periods between patients and controls in F3 channel

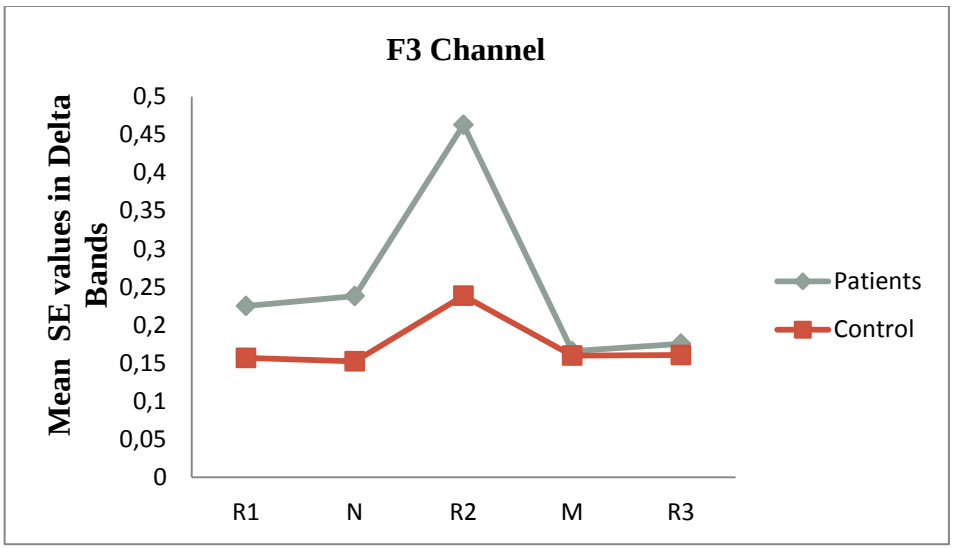
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,156860±0,025671	-0,225090±0,228862	0,361
Delta	N	-0,152430±0,015918	-0,238120±0,267062	0,325
Delta	R2	-0,238540±0,140543	-0,462890±0,301840	0,053*
Delta	M	-0,162840±0,020002	-0,165570±0,043453	0,859
Delta	R3	-0,160700±0,019515	-0,175570±0,053672	0,247

*p≤ 0, 05 is accepted for significant difference

Table 4.3 shows that the significant difference is at R2 state for delta band in F3 region



(a)



(b)

Figure 4.3 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of F3 channel in patient and control groups

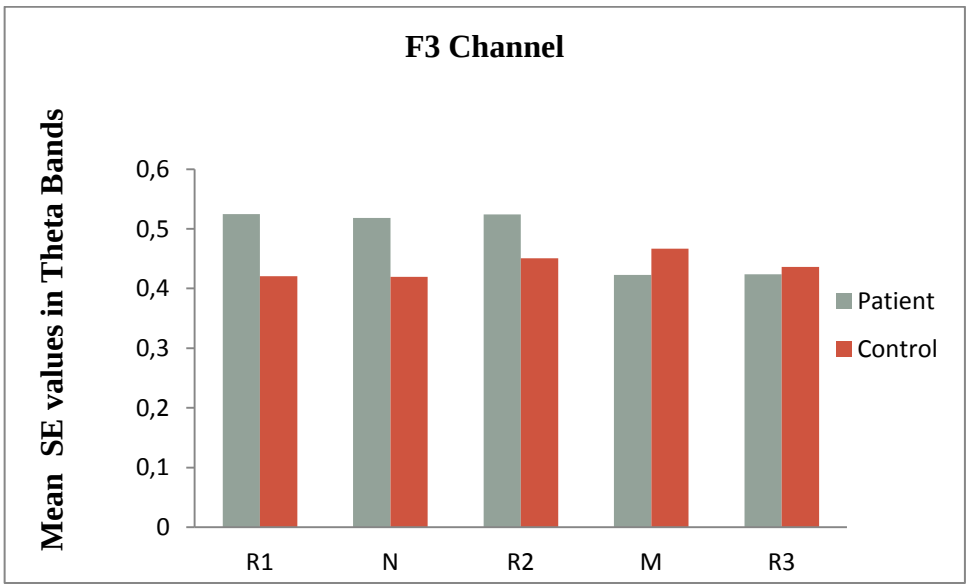
Figure 4.3 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at R1, N, and R2 periods. In M and R3 periods, there is no significant difference between control and patients groups. The mean of SE of groups are almost the same. The mean of SE of patients is highly increasing at R2 state.

Table 4.4 Comparison of SE and p values of Theta band during all measurement periods between patients and controls in F3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,420650±0,035741	-0,524890±0,166075	0,068*
Theta	N	-0,419480±0,035127	-0,518080±0,169886	0,089
Theta	R2	-0,450820±0,058354	-0,524130±0,172372	0,219
Theta	M	-0,466950±0,067309	-0,422630±0,105000	0,276
Theta	R3	-0,436310±0,041743	-0,423800±0,106115	0,733

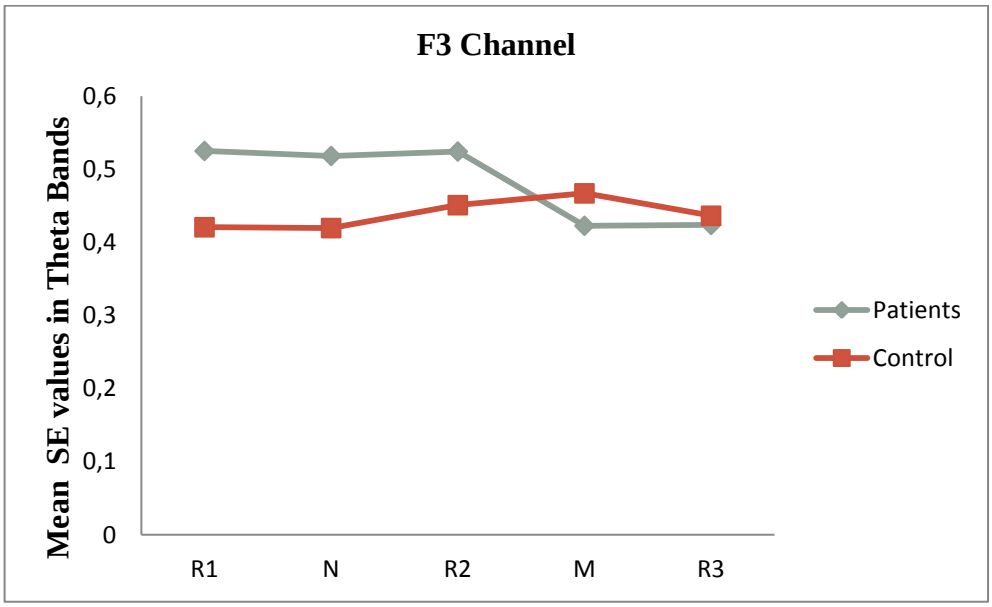
*p≤ 0, 05 is accepted for significant difference. p ≤ 0, 08 is accepted closest value for significant difference

Table 4.4 shows that the significant difference is at R1 state for theta band in F3 channel.



(a)

Figure 4.4 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of F3 channel in patient and control groups



(b)

Figure 4.4 (Continue) (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of F3 channel in patient and control groups

Figure 4.4 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at R1, N, and R2 periods. In M and R3 periods, means of SE of patients are lower than controls.While groups are listening the relaxing music, the mean of SE of controls is increasing.

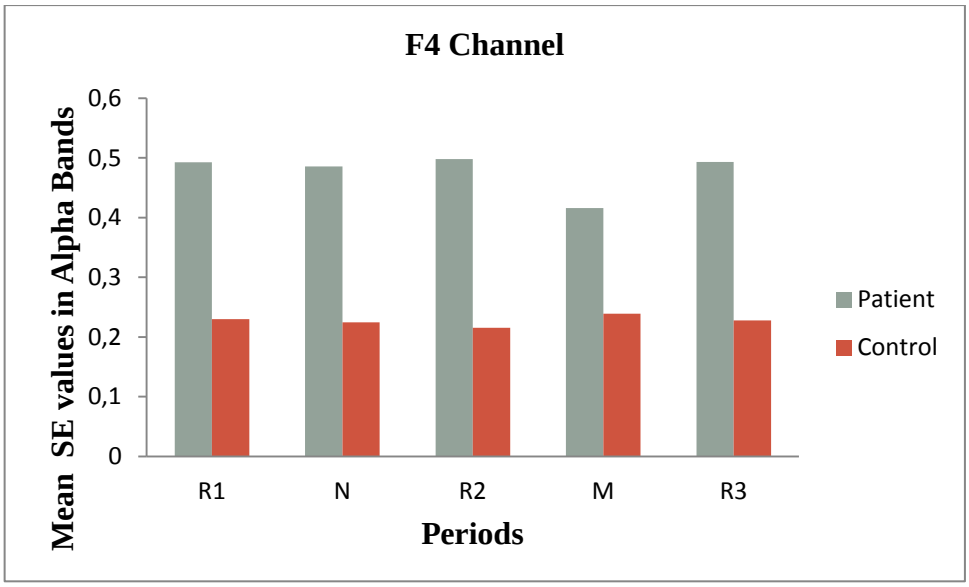
Table 4.5, 4.6, 4.7, and 4.8 show calculated SE and p values in each periods (R1, N, R2, M, R3) between patients and controls in each sub bands respectively alpha, beta, delta, theta in F4 region.

Table 4.5 Comparison of SE and p values of alpha band during all measurement periods between patients and controls in F4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,445760±0,024343	-0,563840±0,164631	0,050*
Alpha	N	-0,462640±0,084292	-0,559780±0,201770	0,177
Alpha	R2	-0,444470±0,019066	-0,590240±0,212843	0,059*
Alpha	M	-0,467080±0,049508	-0,468390±0,128620	0,976
Alpha	R3	-0,459180±0,065145	-0,531170±0,182147	0,264

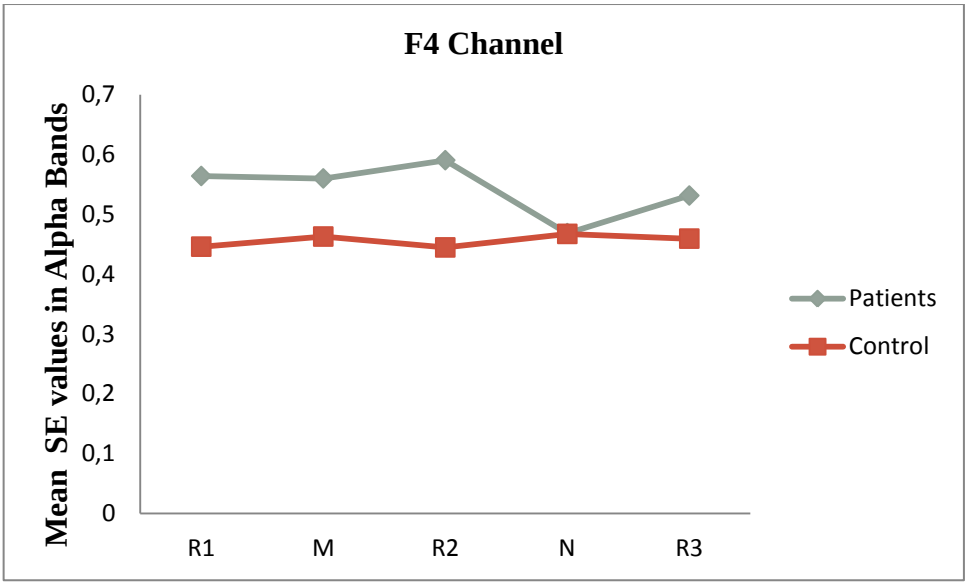
*p≤ 0, 05 is accepted for significant difference

Table 4.5 shows that the significant difference is at R1 and R2 states for alpha band in F4 channel.



(a)

Figure 4.5 (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of F3 channel in patient and control groups



(b)

Figure 4.5 (Continue) (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of F3 channel in patient and control groups

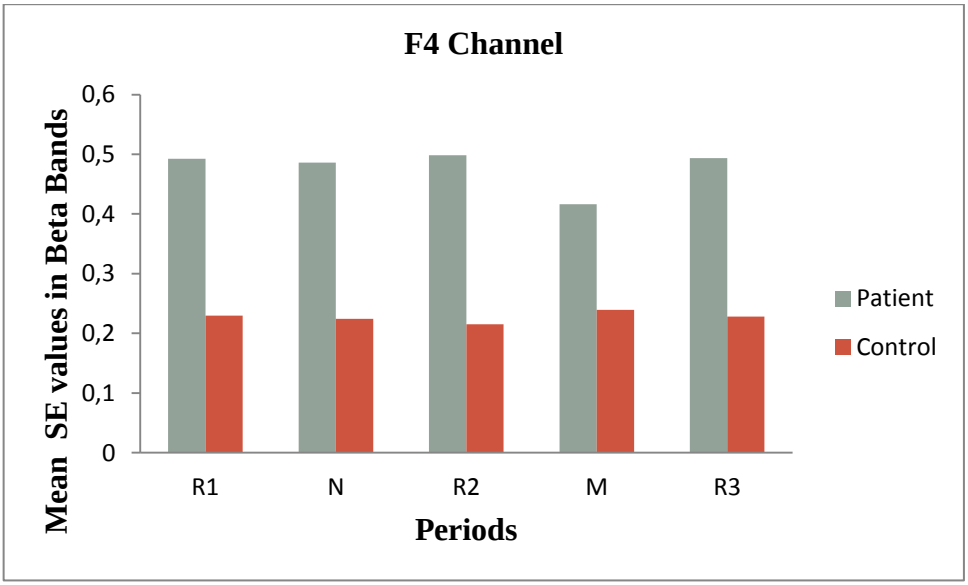
Figure 4.5 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, M, R2, and R3 periods. After R2 state, the mean of SE of patients decrease at M period while the mean of SE of controls is increasing at M state. In N state, the mean of SE of patients is increasing and the mean of SE of controls is decreasing. At the same time, control's mean of SE doesn't change in large at all periods.

Table 4.6 Comparison of SE and p values of beta band during all measurement periods between patients and controls in F4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,229600±0,080996	-0,492600±0,249299	0,009*
Beta	N	-0,224420±0,080846	-0,485920±0,273542	0,015*
Beta	R2	-0,215410±0,063412	-0,498120±0,272355	0,010*
Beta	M	-0,239070±0,107614	-0,416170±0,200202	0,024*
Beta	R3	-0,227970±0,078425	-0,493360±0,278207	0,015*

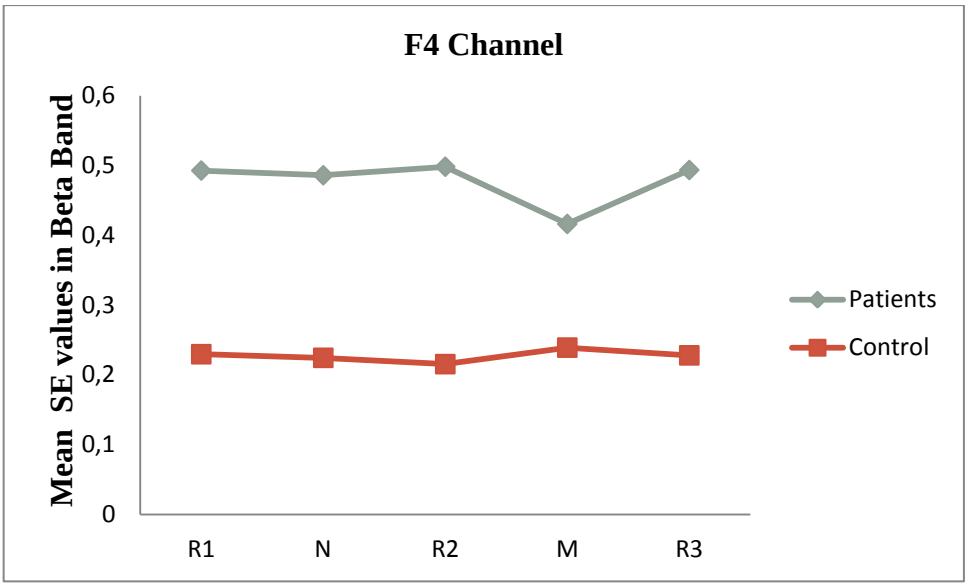
*p≤ 0, 05 is accepted for significant difference

Table 4.6 shows that the significant difference is at all states for beta band in F4 channel.



(a)

Figure 4.6 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of F4 channel in patient and control groups



(b)

Figure 4.6 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of F4 channel in patient and control groups

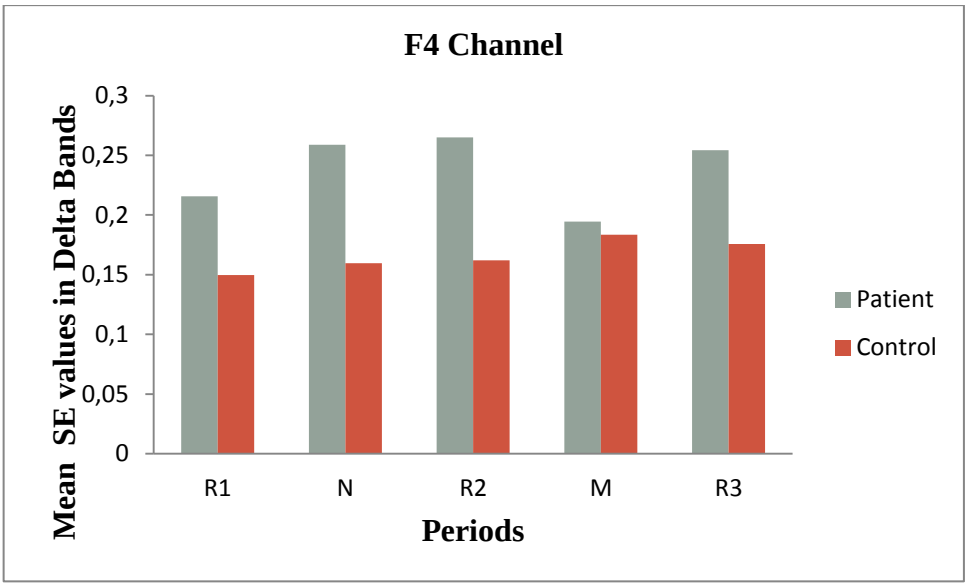
Figure 4.6 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in all periods for beta band in F4 channel. The mean of SE of patients is decreasing at M periods. After M state, the mean of SE of patients is increasing in F4 channel.

Table 4.7 Comparison of SE and p values of delta band during all measurement periods between patients and controls in F4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,149730±0,015316	-0,215770±0,145183	0,186
Delta	N	-0,159650±0,019167	-0,258967±0,250417	0,269
Delta	R2	-0,162110±0,031726	-0,265010±0,256630	0,239
Delta	M	-0,183580±0,057933	-0,194430±0,092074	0,756
Delta	R3	-0,175820±0,049806	-0,254210±0,264249	0,369

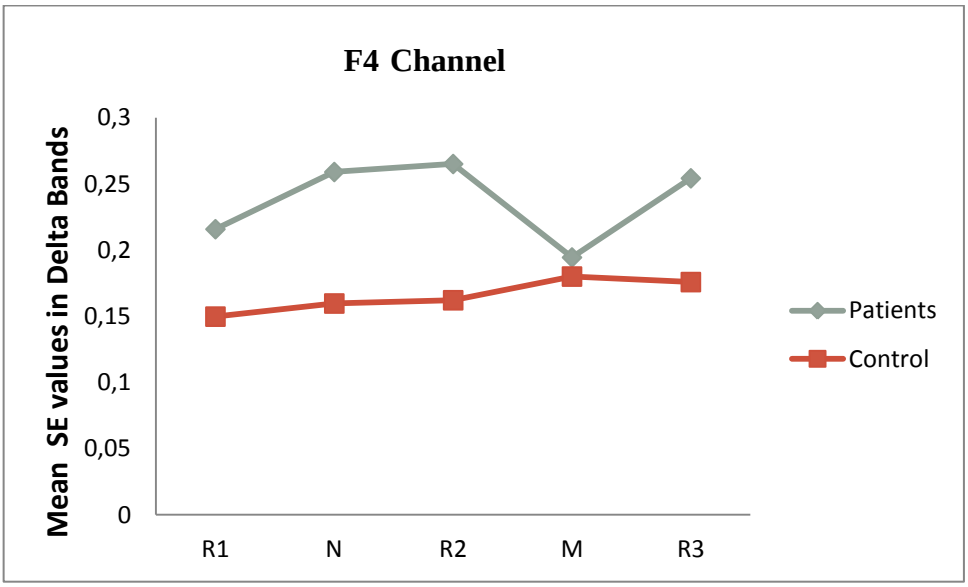
*p≤ 0, 05 is accepted for significant difference

Table 4.7 shows that there is no significant difference at all states for delta band in F4 channel.



(a)

Figure 4.7 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of F4 channel in patient and control groups



(b)

Figure 4.7 (Continue) (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of F4 channel in patient and control groups

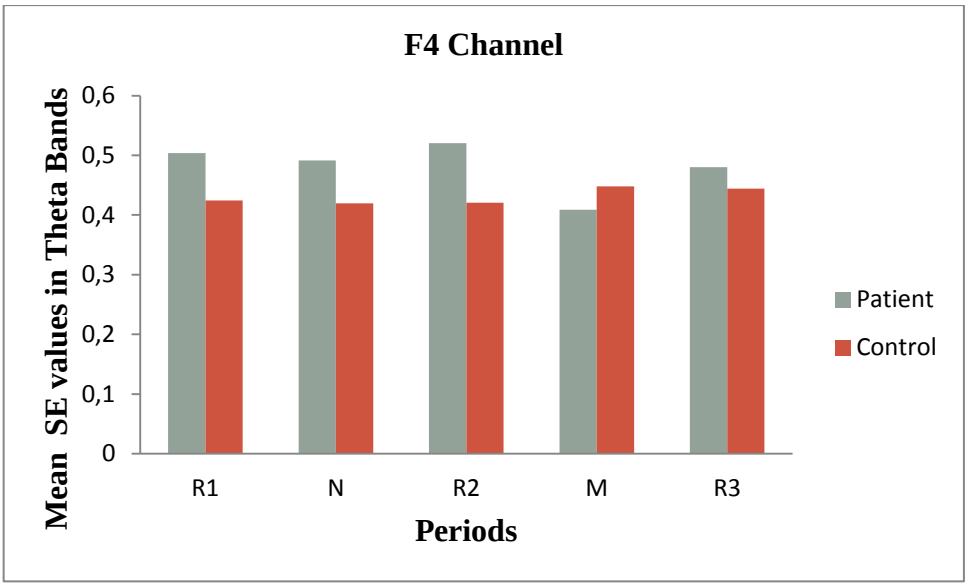
Figure 4.7 shows that t while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, R2, and R3 periods between patients and control groups. There is a little difference in M periods between groups. After R2 state, The mean of SE of patients is decreasing.

Table 4.8 Comparison of SE and p values of theta band during all measurement periods between patients and controls in F4 channel

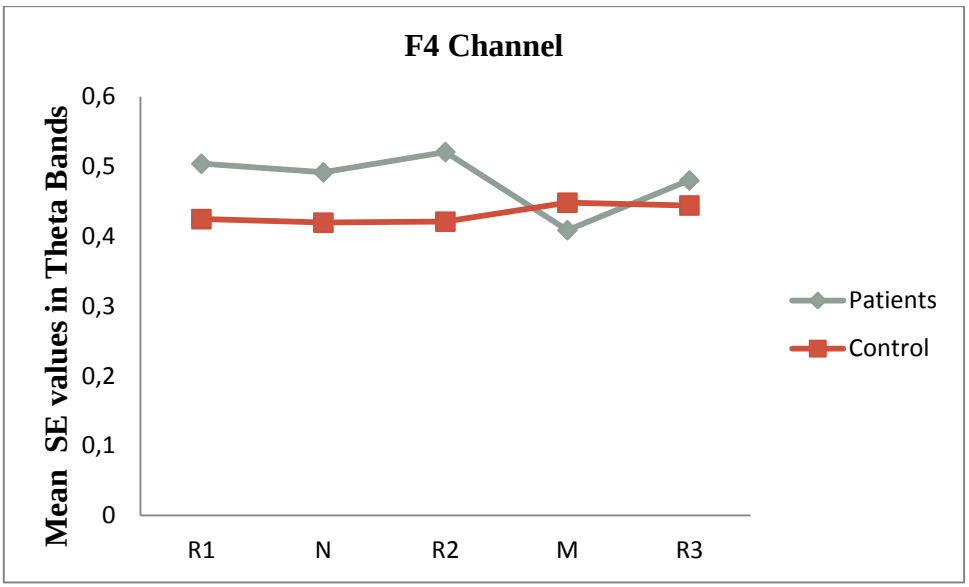
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,424520±0,037233	-0,503820±0,180883	0,191
Theta	N	-0,419430±0,038523	-0,491550±0,175971	0,222
Theta	R2	-0,420870±0,035169	-0,520580±0,185803	0,128
Theta	M	-0,448040±0,056542	-0,408760±0,100413	0,295
Theta	R3	-0,444090±0,032367	-0,479950±0,187746	0,559

*p≤ 0, 05 is accepted for significant difference

Table 4.8 shows that there is no significant difference at all states for theta band in F4 channel.



(a)



(b)

Figure 4.8 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of F4 channel in patient and control groups

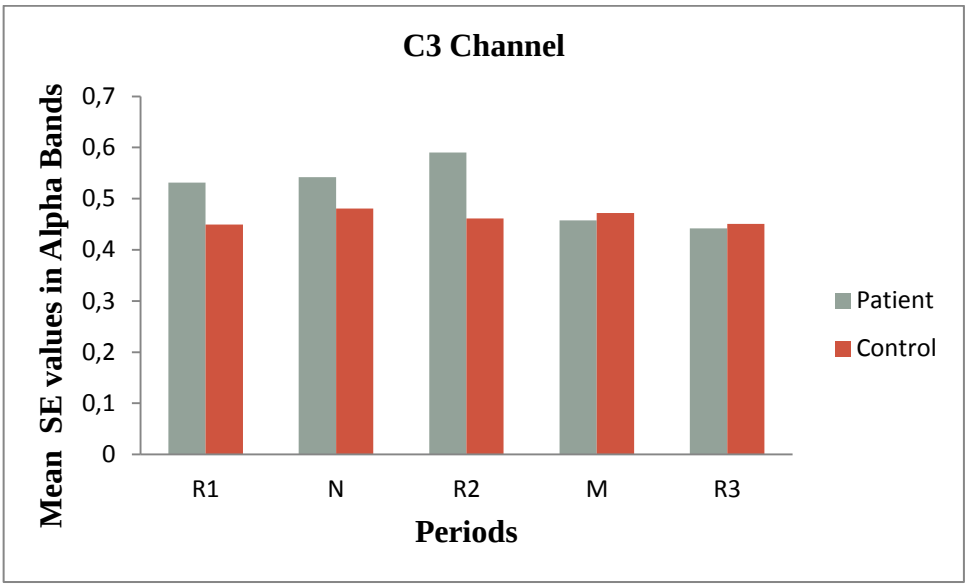
Figure 4.8 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, R2, and R3 periods between patients and control groups. In M period, mean of SE of patients is lower than SE value of controls. At M period, the mean of SE value of patients is decreasing.

Table 4.9 Comparison of SE and p values of alpha band during all measurement periods between patients and controls in C3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,449500±0,031988	-0,531540±0,163590	0,163
Alpha	N	-0,480450±0,069192	-0,541870±0,183991	0,184
Alpha	R2	-0,461400±0,049188	-0,590310±0,206334	0,206
Alpha	M	-0,472090±0,046337	-0,457710±0,138535	0,139
Alpha	R3	-0,450430±0,055822	-0,441680±0,116572	0,116

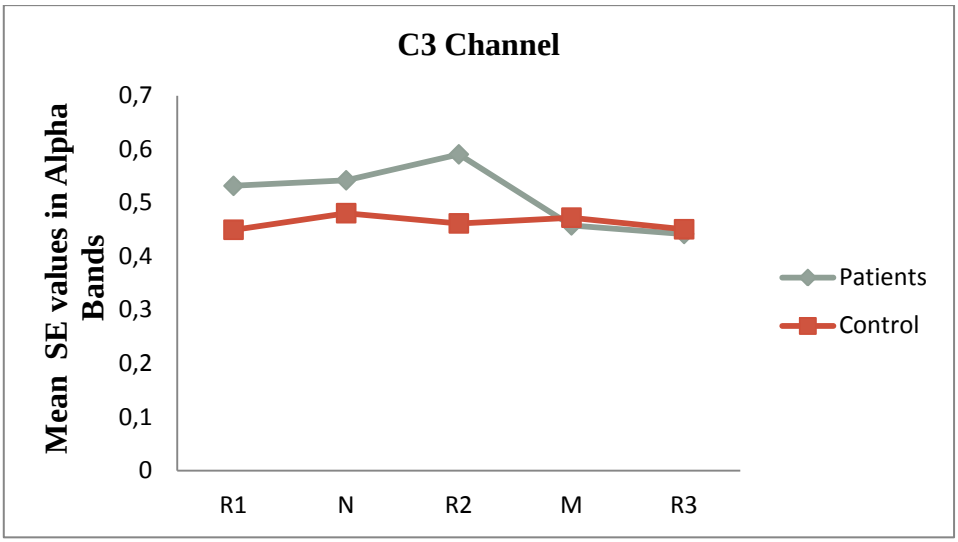
*p≤ 0, 05 is accepted for significant difference

Table 4.9 shows that there is no significant difference at all states for alpha band in C3 channel.



(a)

Figure 4.9 (Continue) (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of C3 channel in patient and control groups



(b)

Figure 4.9 (Continue) (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of C3 channel in patient and control groups

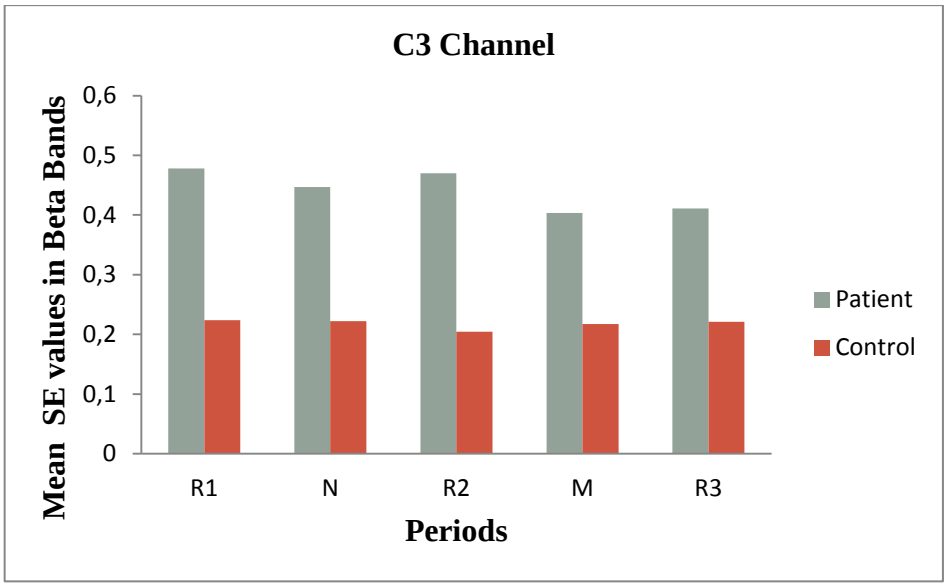
Figure 4.9 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, and R2 periods. In R2 period, the difference is higher than other periods. There is a little difference at M and R3 periods between groups.

Table 4.10 Comparison of SE and p values of beta band during all measurement periods between patients and controls in C3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,223510±0,062100	-0,478180±0,215157	0,005*
Beta	N	-0,222090±0,073003	-0,447010±0,219834	0,007*
Beta	R2	-0,204580±0,045169	-0,470110±0,255498	0,009*
Beta	M	-0,217560±0,056692	-0,403380±0,207214	0,014*
Beta	R3	-0,220950±0,070823	-0,410880±0,225583	0,021*

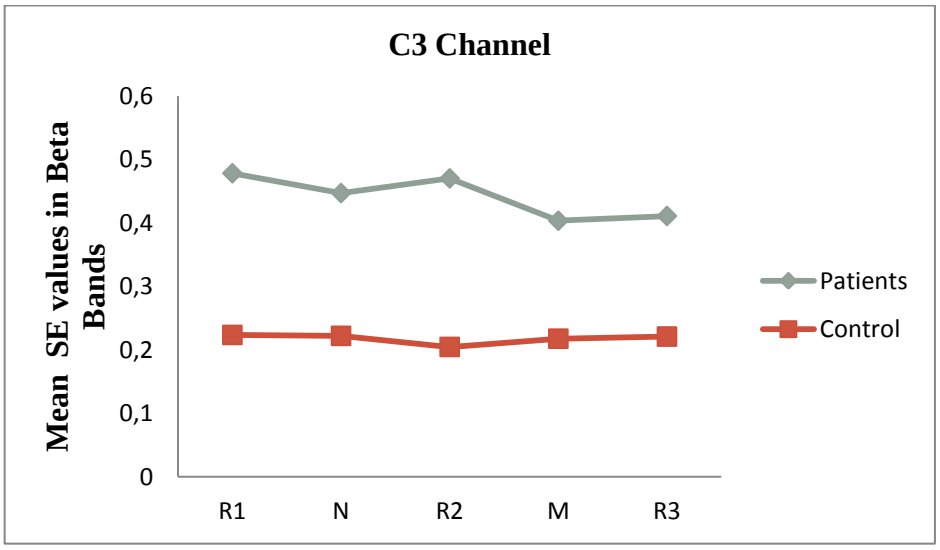
*p≤ 0, 05 is accepted for significant difference

Table 4.10 shows that the significant difference is at all states for beta band in C3 channel.



(a)

Figure 4.10 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of C3 channel in patient and control groups



(b)

Figure 4.10 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of C3 channel in patient and control groups

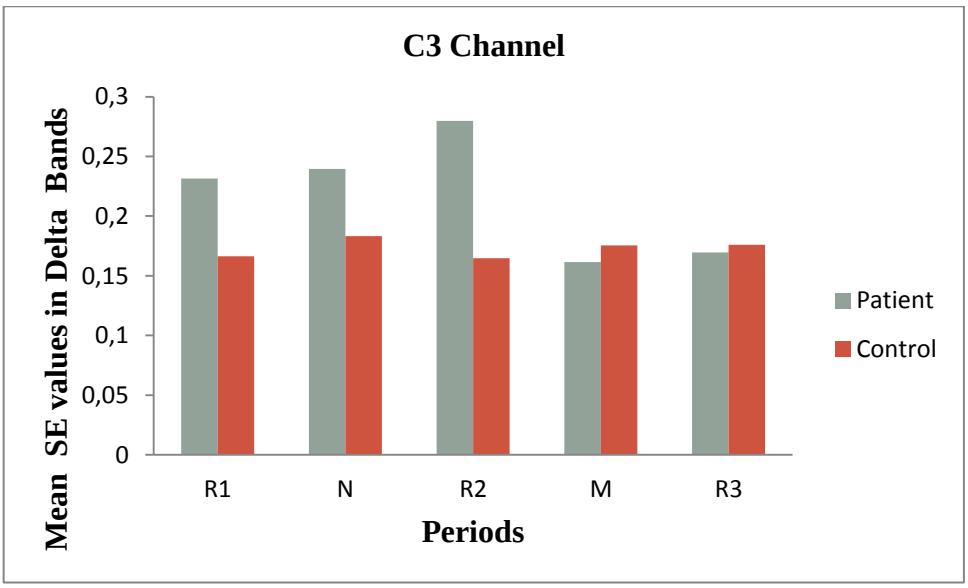
Figure 4.10 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at all channel between groups. The mean of SE values of patients is decreasing at M state.

Table 4.11 Comparison of SE and p values of Delta bandduring all measurement periods between patients and controls in C3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,166430±0,036559	-0,231480±0,183667	0,298
Delta	N	-0,183180±0,060377	-0,239520±0,240530	0,482
Delta	R2	-0,164640±0,030728	-0,279640±0,273668	0,218
Delta	M	-0,175500±0,047137	-0,161370±0,044196	0,498
Delta	R3	-0,176030±0,041325	-0,169460±0,050396	0,754

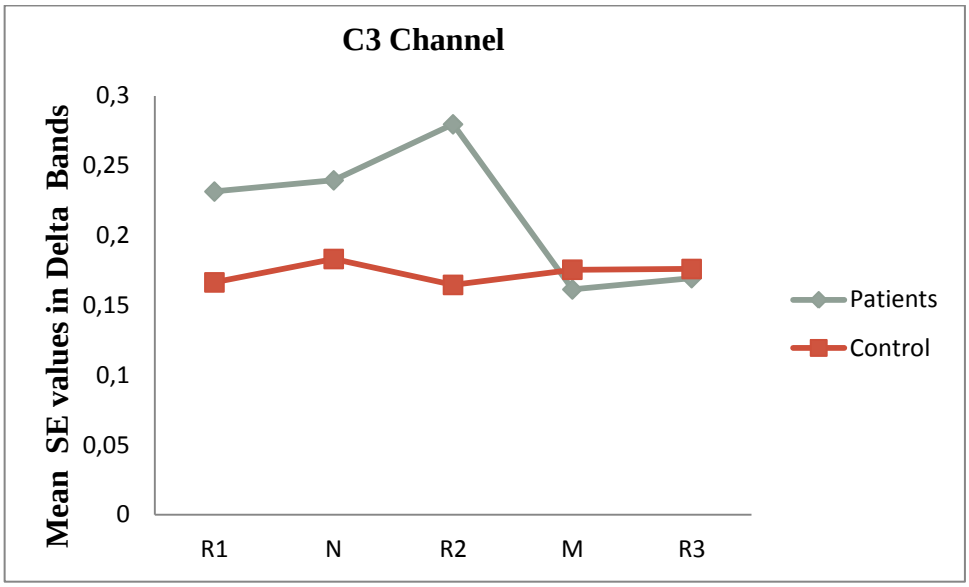
*p≤ 0, 05 is accepted for significant difference

Table 4.11 shows that there is no significant difference at all states for delta band in C3 channel.



(a)

Figure 4.11 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of C3 channel in patient and control groups



(b)

Figure 4.11 (Continue) (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of C3 channel in patient and control groups

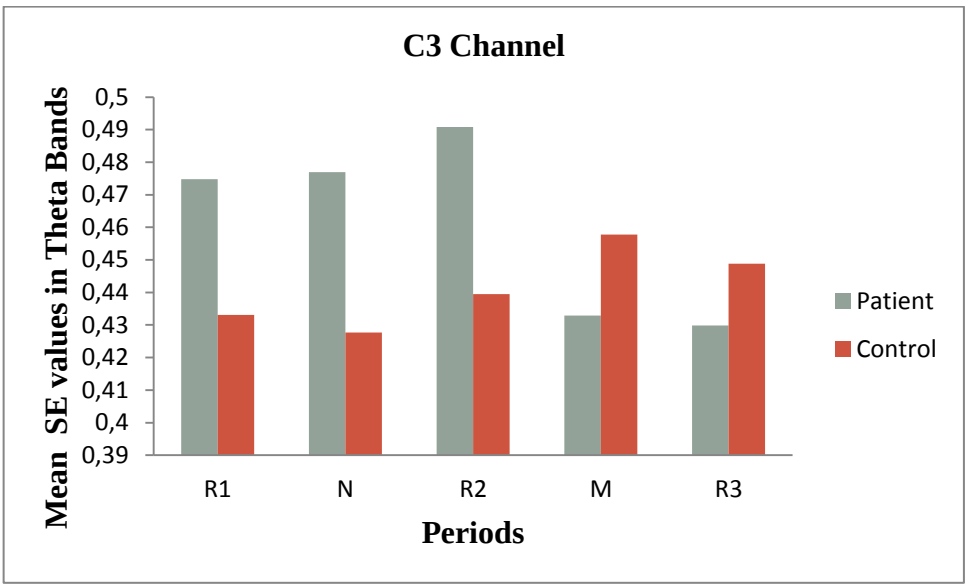
Figure 4.11 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N and R2 periods. The significant difference is shown at R2 period. The mean of SE values of patients is decreasing at M state.

Table 4.12 Comparison of SE and p values of Theta band during all measurement periods between patients and controls in C3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,433050±0,048065	-0,474830±0,204157	0,537
Theta	N	-0,427710±0,041117	-0,476930±0,203971	0,464
Theta	R2	-0,439470±0,040157	-0,490830±0,215968	0,469
Theta	M	-0,457760±0,051716	-0,432910±0,107231	0,518
Theta	R3	-0,448830±0,048965	-0,429830±0,107510	0,617

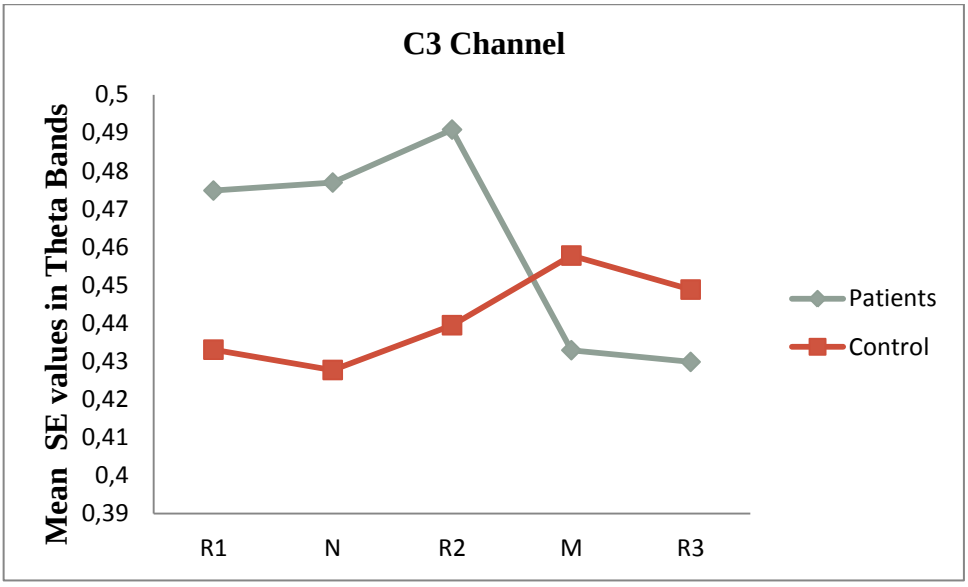
*p≤ 0, 05 is accepted for significant difference

Table 4.12 shows that there is no significant difference at all states for theta band in C3 channel



(a)

Figure 4.12 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of C3 channel in patient and control groups



(b)

Figure 4.12 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of C3 channel in patient and control groups

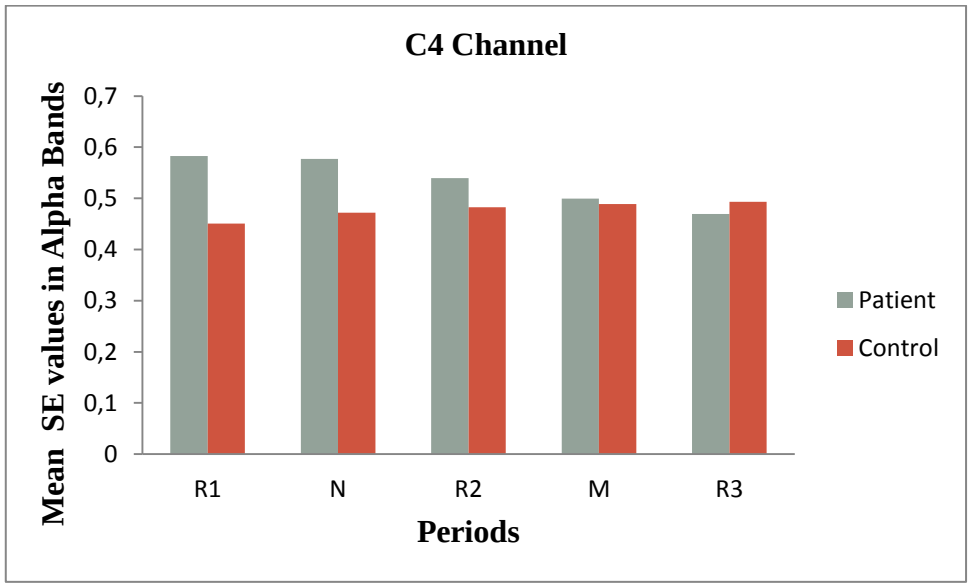
Figure 4.12 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE value of patients is higher than controls in R1, N, and R2 periods. The mean of SE value of patients is increasing from R1 to R2 periods. The mean of SE value of patients are decreasing M and R3 periods.

Table 4.13 Comparison of SE and p values of Alpha band during all measurement periods between patients and controls in C4 channel

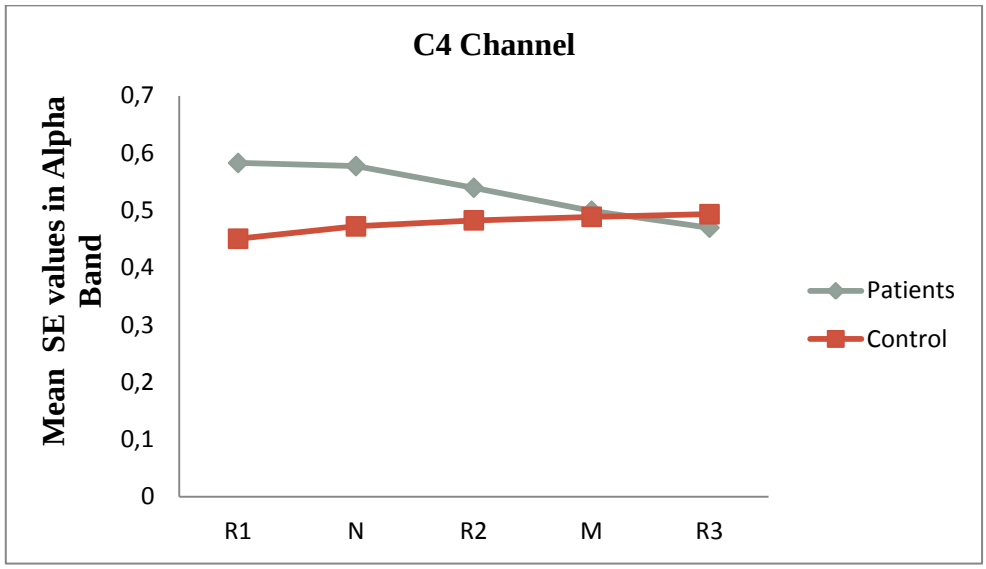
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,450480±0,056659	-0,582710±0,198961	0,070*
Alpha	N	-0,472020±0,063873	-0,577320±0,186858	0,120
Alpha	R2	-0,482590±0,076652	-0,539410±0,178513	0,367
Alpha	M	-0,488690±0,078960	-0,499340±0,156474	0,850
Alpha	R3	-0,493310±0,086801	-0,469690±0,159909	0,686

*p≤ 0, 05 is accepted for significant difference. p ≤ 0, 08 is accepted closest significant difference

Table 4.13 shows that the significant difference is at R1 state for alpha band in C4 channel.



(a)



(b)

Figure 4.13 (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of C4 channel in patient and control groups

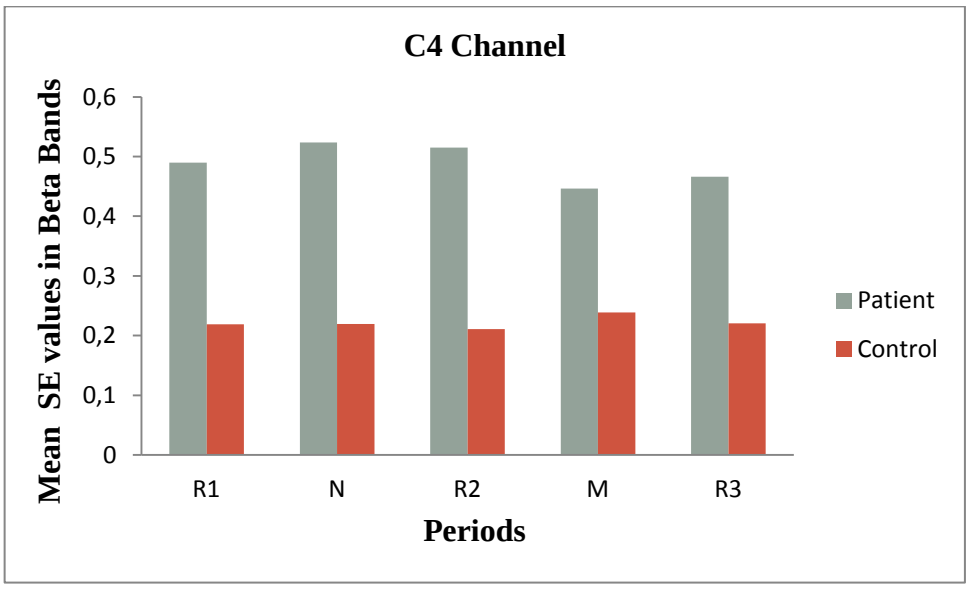
Figure 4.13 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, R2, and M periods. There is no significant difference between periods. The mean of SE value of patiens is decreasing at M period.

Table 4.14 Comparison of SE and p values of Beta band during all measurement periods between patients and controls in C4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,219120±0,077655	-0,489530±0,276130	0,013*
Beta	N	-0,219260±0,076411	-0,523410±0,271522	0,006*
Beta	R2	-0,210820±0,062200	-0,514860±0,287866	0,009*
Beta	M	-0,238810±0,117785	-0,446460±0,220212	0,017*
Beta	R3	-0,220670±0,076284	-0,466230±0,220068	0,007*

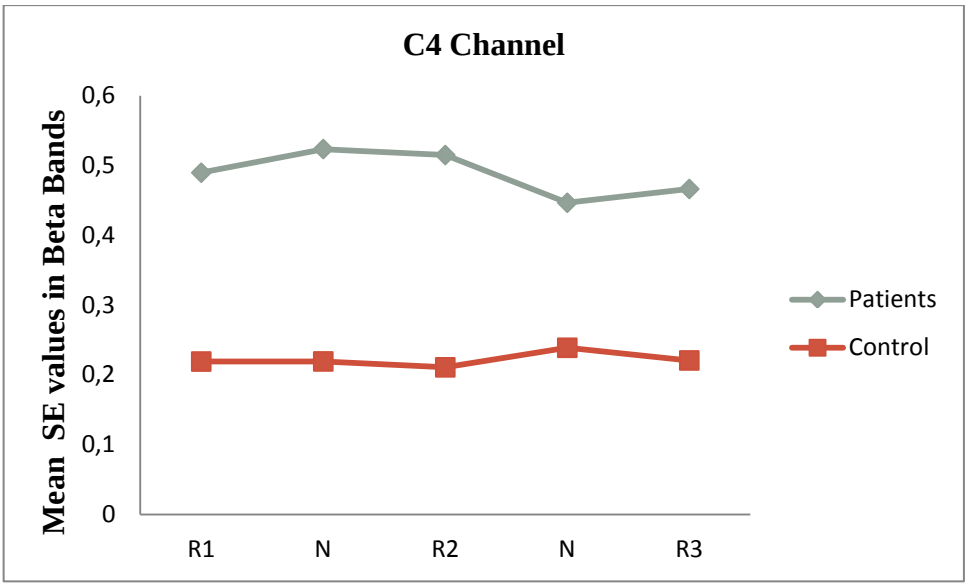
*p≤ 0, 05 is accepted for significant difference

Table 4.14 shows that the significant difference is at all states for beta band in C4 channel.



(a)

Figure 4.14 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of C4 channel in patient and control groups



(b)

Figure 4.14 (Continue) (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of C4 channel in patient and control groups

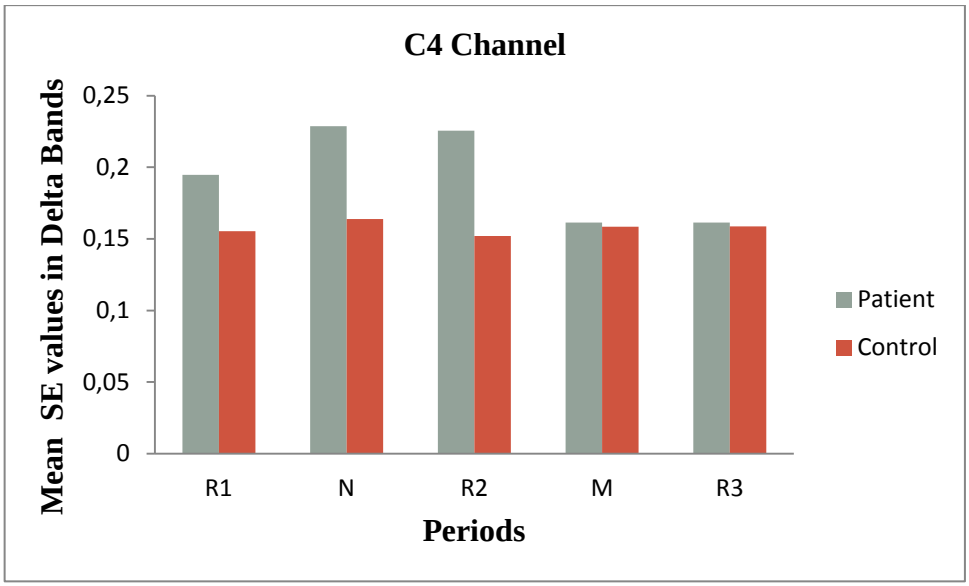
Figure 4.14 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in all periods. The mean of SE values of patients is decresing after R2 periods. It is again increasing at R3 period.

Table 4.15 Comparison of SE and p values of Delta band during all measurement periods between patients and controls in C4 channel

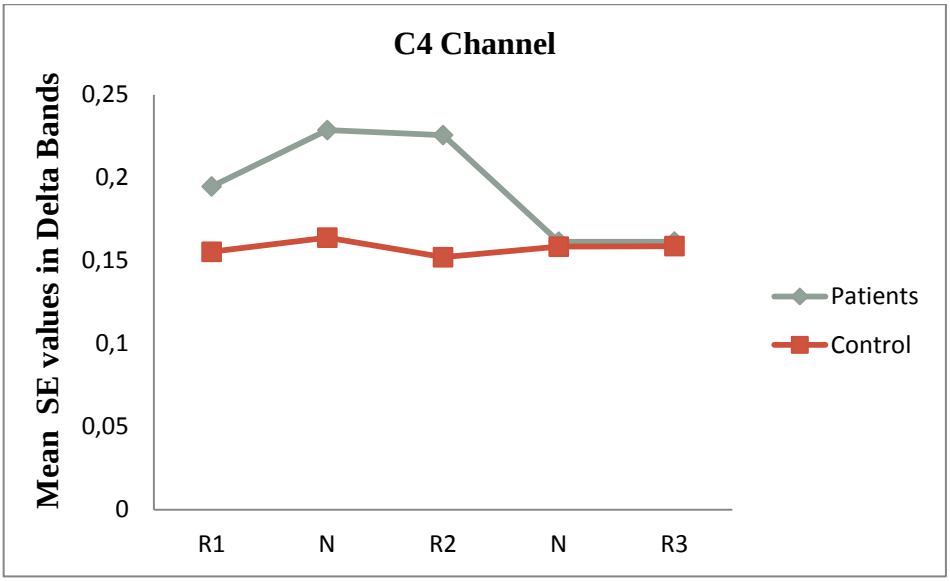
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,155310±0,027111	-0,194690±0,134740	0,377
Delta	N	-0,163840±0,024550	-0,228640±0,250105	0,426
Delta	R2	-0,152030±0,016182	-0,225620±0,245929	0,358
Delta	M	-0,158410±0,019471	-0,161410±0,044369	0,847
Delta	R3	-0,158760±0,020577	-0,161470±0,044623	0,863

*p≤ 0, 05 is accepted for significant difference

Table 4.15 shows that there is no significant difference at all channels.



(a)



(b)

Figure 4.15 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of C4 channel in patient and control groups

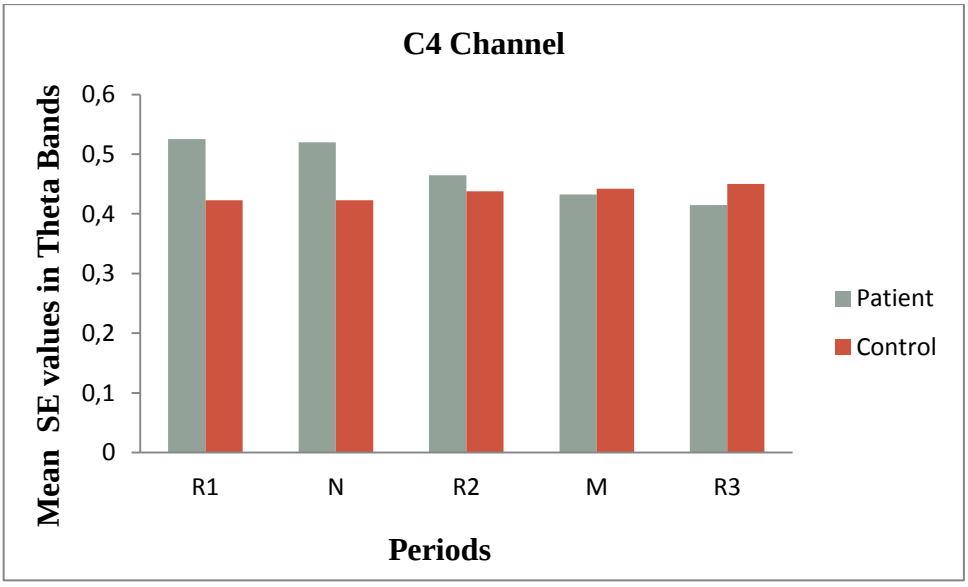
Figure 4.15 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing M period.

Table 4.16 Comparison of SE and p values of theta band during all measurement periods between patients and controls in C4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,422860±0,048175	-0,525090±0,154466	0,061*
Theta	N	-0,422520±0,040759	-0,519690±0,167444	0,091
Theta	R2	-0,437730±0,058318	-0,464725±0,064445	0,365
Theta	M	-0,442030±0,058664	-0,432160±0,103173	0,796
Theta	R3	-0,450350±0,063935	-0,414620±0,097775	0,346

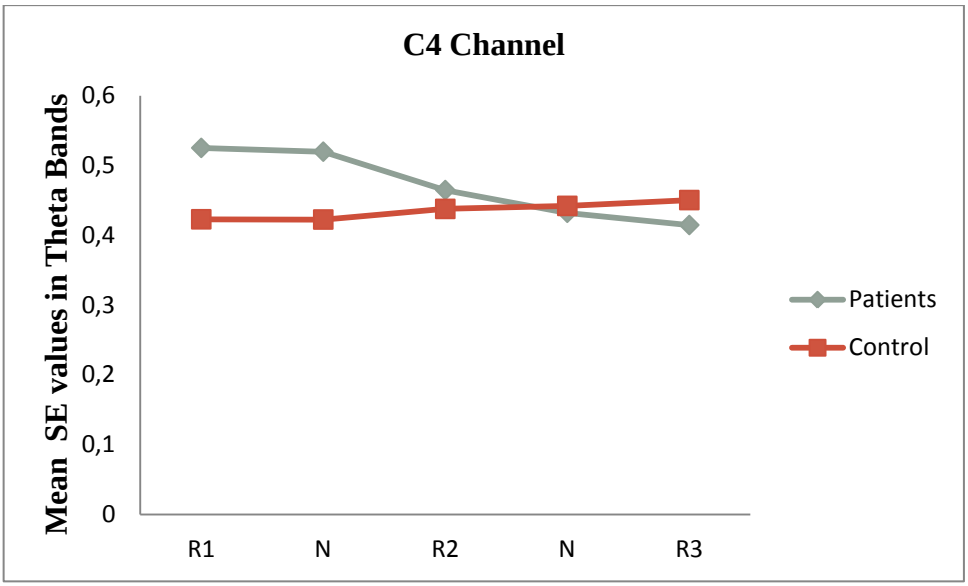
*p≤ 0, 05 is accepted for significant difference

Table 4.16 shows that the significant difference is at R1 state for theta band in C4 channel.



(a)

Figure 4.16 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of C4 channel in patient and control groups



(b)

Figure 4.16 (Continue) (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of C4 channel in patient and control groups

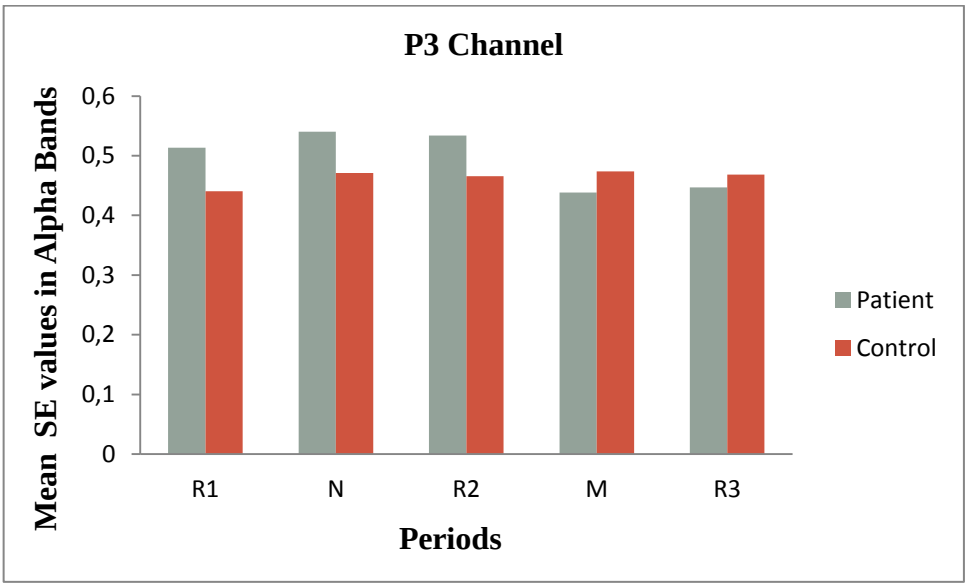
Figure 4.16 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE values of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing after R2 periods.

Table 4.17 Comparison of SE and p values of alpha band during all measurement periods between patients and controls in P3 channel

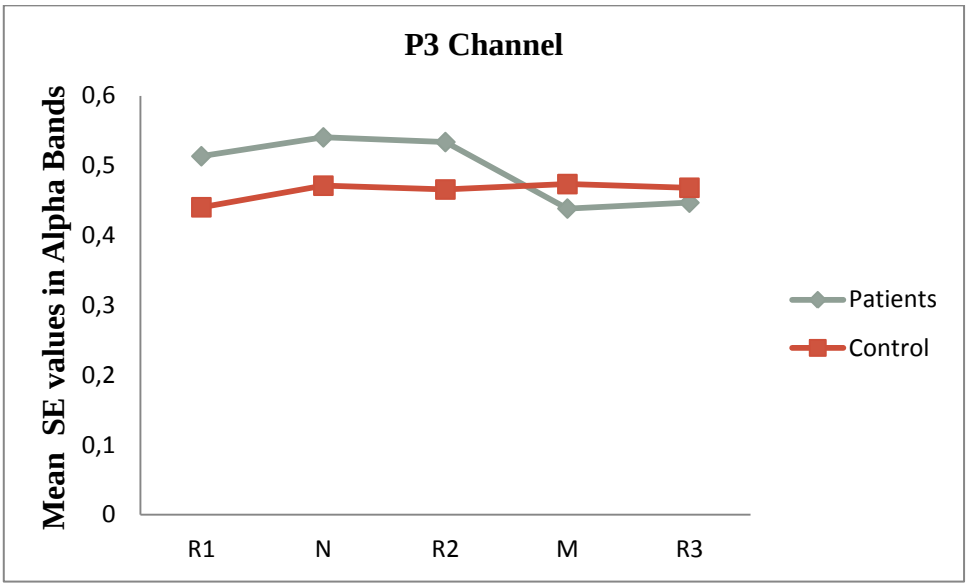
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,440340±0,028934	-0,513410±0,172616	0,203
Alpha	N	-0,471250±0,038692	-0,540420±0,174582	0,250
Alpha	R2	-0,465800±0,043232	-0,533720±0,169224	0,235
Alpha	M	-0,473550±0,037729	-0,438500±0,108938	0,349
Alpha	R3	-0,468270±0,045435	-0,446900±0,112901	0,586

*p≤ 0, 05 is accepted for significant difference

Table 4.17 shows that there is no significant difference at all states for alpha band in P3 channel.



(a)



(b)

Figure 4.17 (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of P3 channel in patient and control groups

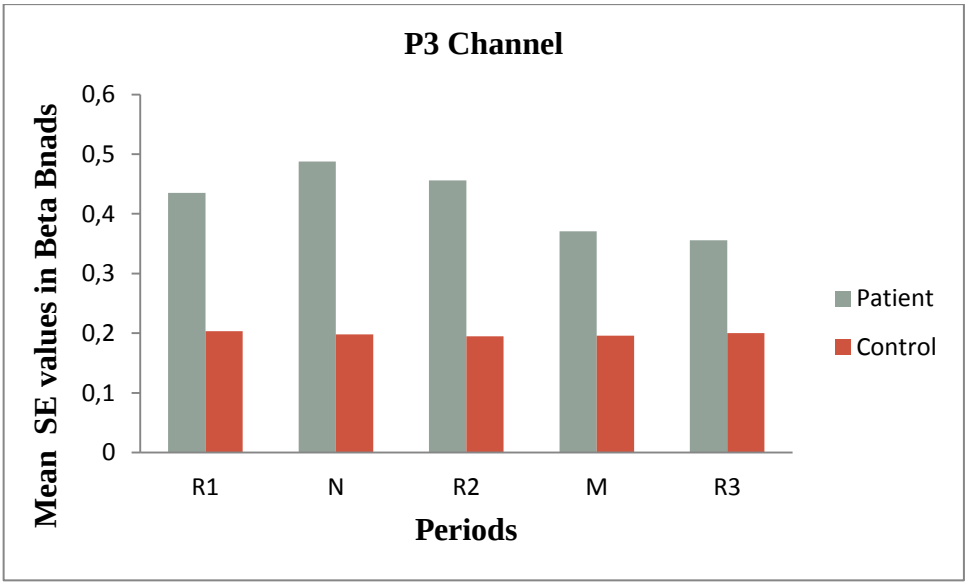
Figure 4.17 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE values of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing at M state.

Table 4.18 Comparison of SE and p values of Beta band during all measurement periods between patients and controls in P3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,203360±0,032362	-0,435110±0,229242	0,011*
Beta	N	-0,198120±0,029304	-0,487560±0,257456	0,006*
Beta	R2	-0,194700±0,033316	-0,456030±0,258382	0,011*
Beta	M	-0,196030±0,031880	-0,370650±0,130203	0,001*
Beta	R3	-0,200260±0,037341	-0,355880±0,140897	0,003*

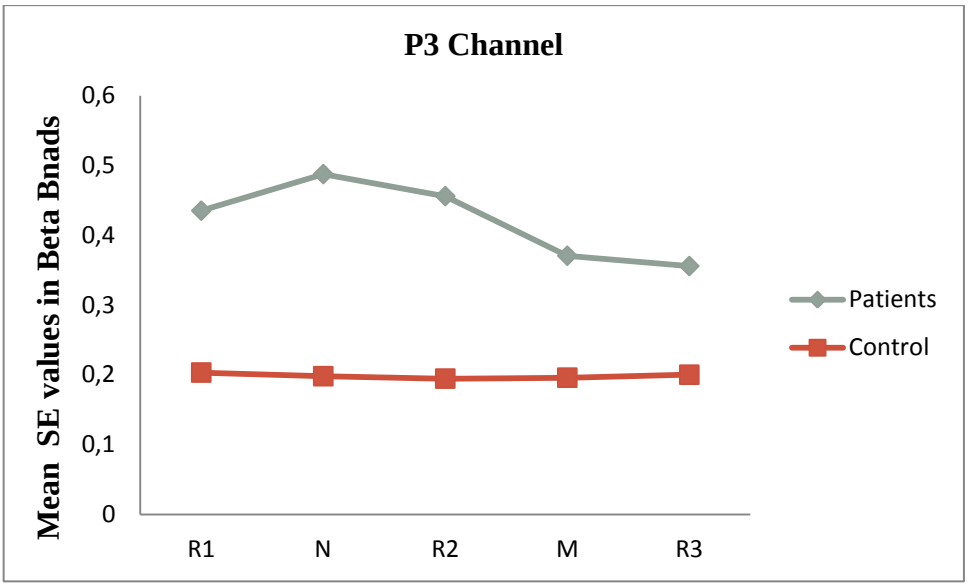
*p≤ 0, 05 is accepted for significant difference

Table 4.18 shows that the significant difference is at all states for beta band in P3 channel.



(a)

Figure 4.18 (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of P3 channel in patient and control groups



(b)

Figure 4.18(Continue) (a) shows that changes in the beta band with column graph and (b) shows that changes in the beta band with line graph of P3 channel in patient and control groups

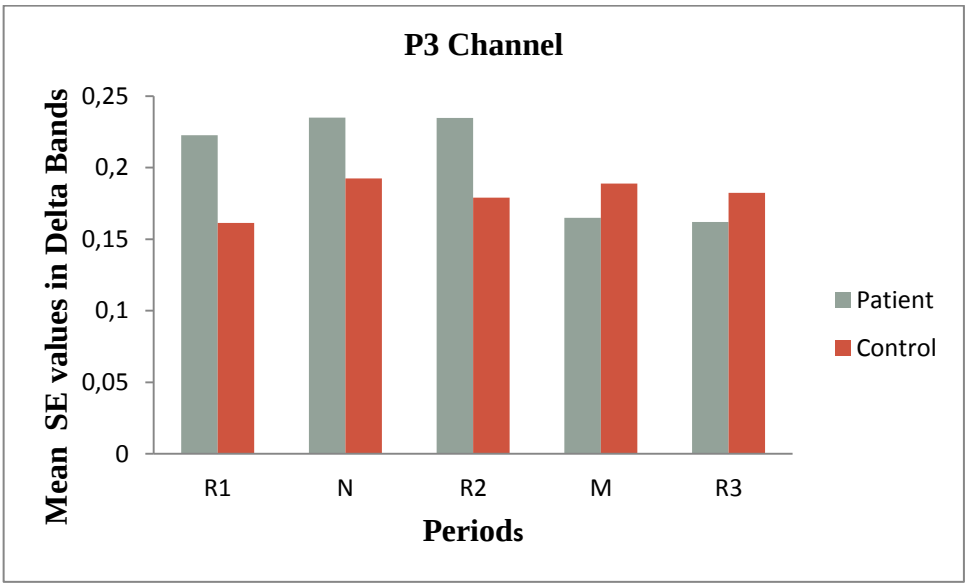
Figure 4.18 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls at all periods. The highest SE value of patients is at N period. During M period, the mean of SE values of patients is decreasing.

Table 4.19 Comparison of SE and p values of Delta band during all measurement periods between patients and controls in P3 channel

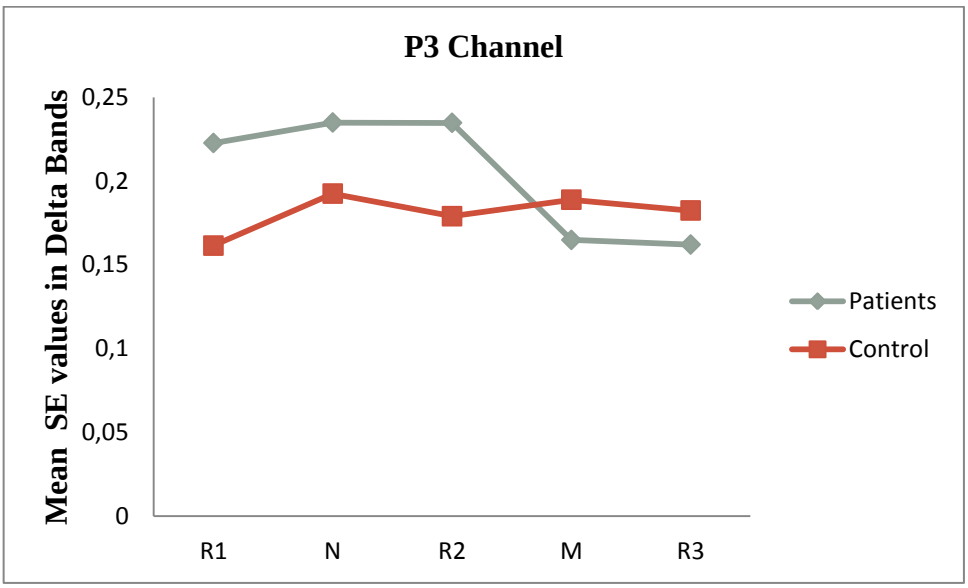
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,161410±0,026589	-0,222670±0,238777	0,431
Delta	N	-0,192450±0,045510	-0,234900±0,254128	0,609
Delta	R2	-0,178960±0,047550	-0,234700±0,266245	0,523
Delta	M	-0,188810±0,044881	-0,164810±0,045942	0,253
Delta	R3	-0,182380±0,054861	-0,162070±0,045279	0,379

*p≤ 0, 05 is accepted for significant difference

Table 4.19 shows that there is no significant difference at all states for delta band in P3 channel.



(a)



(b)

Figure 4.19 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of P3 channel in patient and control groups

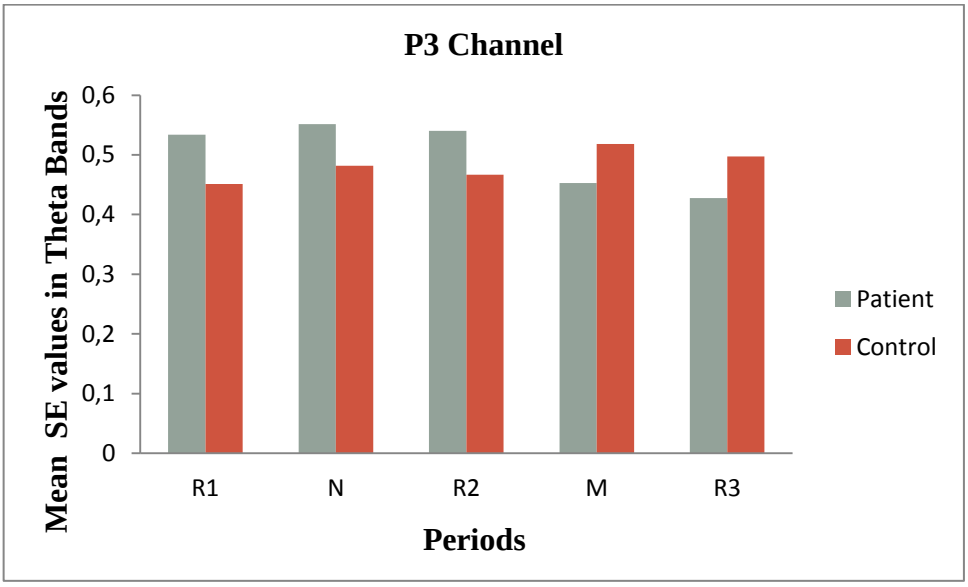
Figure 4.19 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing after R2 periods.

Table 4.20 Comparison of EEG Theta band features during all measurement periods between patients and controls in P3 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,427280±0,064611	-0,498760±0,180052	0,253
Theta	N	-0,433090±0,049211	-0,493180±0,175449	0,311
Theta	R2	-0,441210±0,034978	-0,507740±0,177696	0,261
Theta	M	-0,475470±0,066964	-0,424680±0,106322	0,217
Theta	R3	-0,455620±0,047995	-0,412940±0,108629	0,271

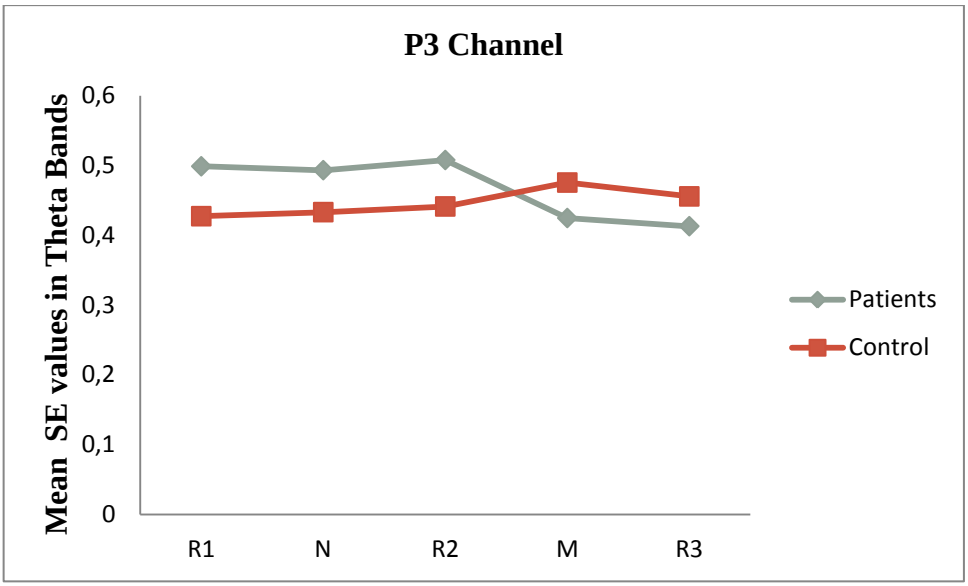
*p≤ 0, 05 is accepted for significant difference

Table 4.20 shows that there is no significant difference at all states for theta band in P3 channel.



(a)

Figure 4.20 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of P3 channel in patient and control groups



b)

Figure 4.20 (Continue) (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of P3 channel in patient and control groups

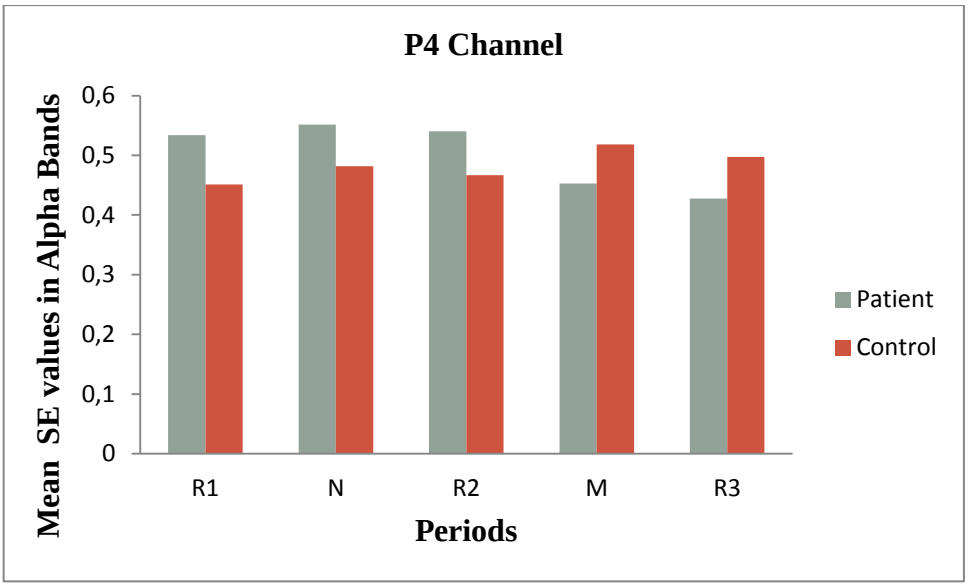
Figure 4.20 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of controls is increasing after R2 periods.The mean of SE values of patients is decreasing at M period.

Table 4.21 Comparison of SE and p values of Alpha band during all measurement periods between patients and controls in P4 channel

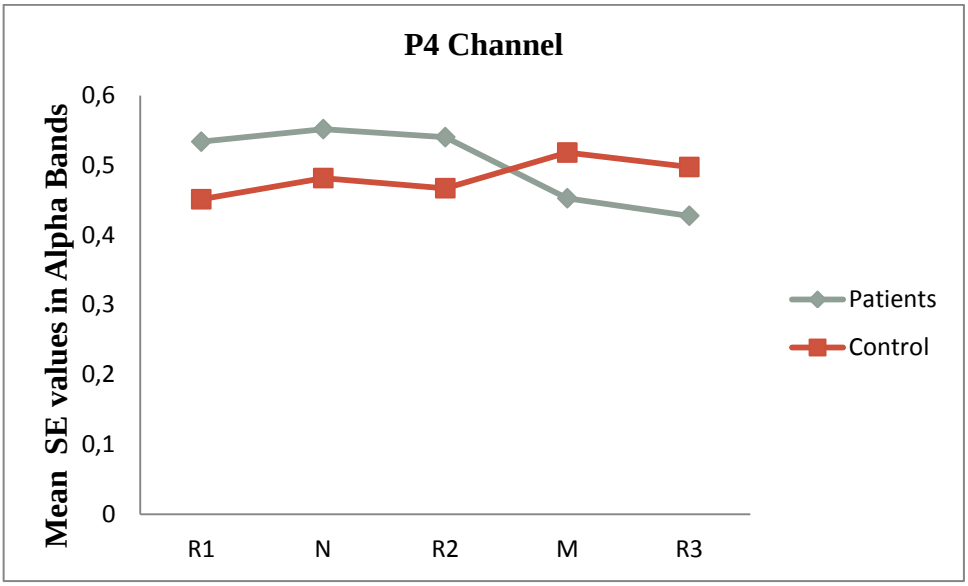
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Alpha	R1	-0,451160±0,039008	-0,533630±0,164473	0,140
Alpha	N	-0,481490±0,069642	-0,551640±0,183367	0,273
Alpha	R2	-0,466980±0,067989	-0,540130±0,190741	0,268
Alpha	M	-0,518050±0,080574	-0,452610±0,122731	0,176
Alpha	R3	-0,497360±0,075029	-0,427550±0,101820	0,980

*p≤ 0, 05 is accepted for significant difference

Table 4.21 shows that there is no significant difference at all states for alpha band in P4 channel.



(a)



(b)

Figure 4.21 (a) shows that changes in the alpha band with column graph and (b) shows that changes in the alpha band with line graph of P4 channel in patient and control groups

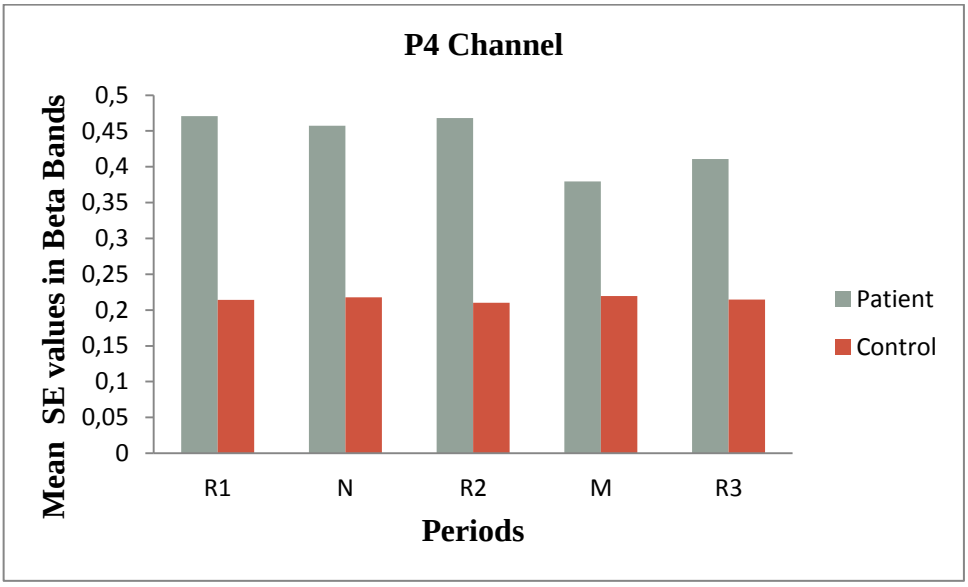
Figure 4.21 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE values of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing after R2 period.

Table 4.22 Comparison of SE and p values of beta band during all measurement periods between patients and controls in P4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Beta	R1	-0,214310±0,072233	-0,470690±0,223563	0,006*
Beta	N	-0,217900±0,071763	-0,457220±0,220448	0,004*
Beta	R2	-0,210050±0,063493	-0,468010±0,241922	0,008*
Beta	M	-0,219380±0,075994	-0,379550±0,106446	0,001*
Beta	R3	-0,214520±0,060784	-0,410800±0,212207	0,012*

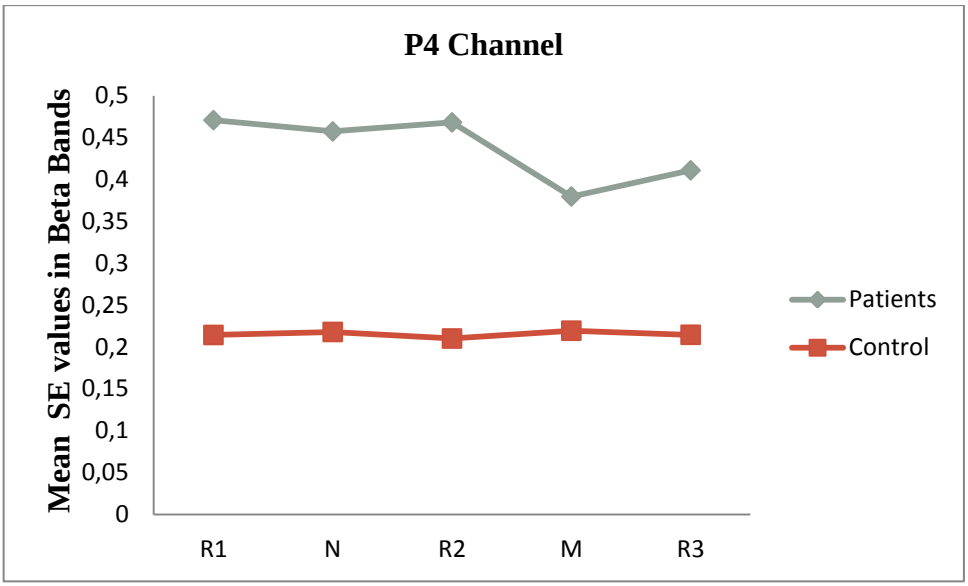
*p≤ 0, 05 is accepted for significant difference

Table 4.22 shows that the significant difference is at all states for beta band in P4 channel.



(a)

Figure 4.22 (a) shows that changes in the belta band with column graph and (b) shows that changes in the beta band with line graph of P4 channel in patient and control groups



(b)

Figure 4.22 (Continue) (a) shows that changes in the belta band with column graph and (b) shows that changes in the beta band with line graph of P4 channel in patient and control groups

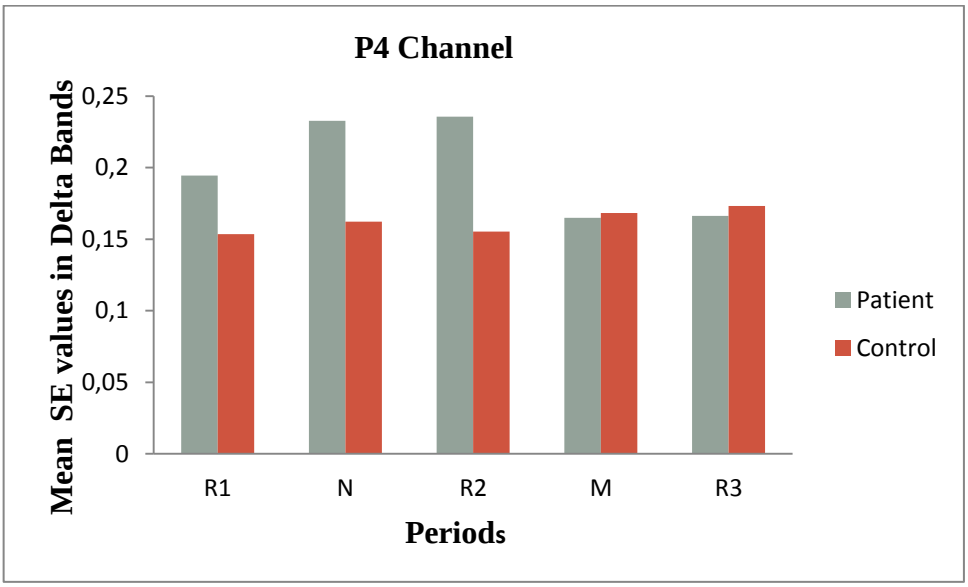
Figure 4.22 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE values of patients is higher than controls at all periods. After M period, the mean of SE values of patients is increasing. The highest mean of SE values of patients is in R2 state.

Table 4.23 Comparison of SE and p values of Delta band during all measurement periods between patients and controls in P4 channel

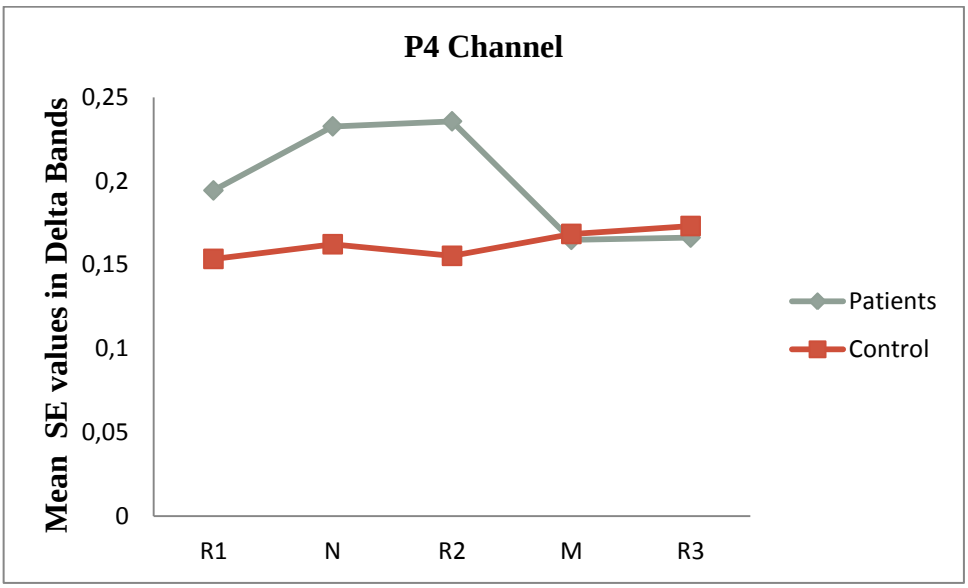
Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Delta	R1	-0,153460±0,017462	-0,194350±0,133534	0,350
Delta	N	-0,162220±0,023339	-0,232580±0,254595	0,396
Delta	R2	-0,155300±0,022588	-0,235600±0,263798	0,350
Delta	M	-0,168370±0,045050	-0,164850±0,042999	0,860
Delta	R3	-0,173090±0,044359	-0,166190±0,044544	0,733

*p≤ 0, 05 is accepted for significant difference

Table 4.23 shows that there is no significant difference at ll1 states for delta band in P4 channel.



(a)



(b)

Figure 4.23 (a) shows that changes in the delta band with column graph and (b) shows that changes in the delta band with line graph of P4 channel in patient and control groups

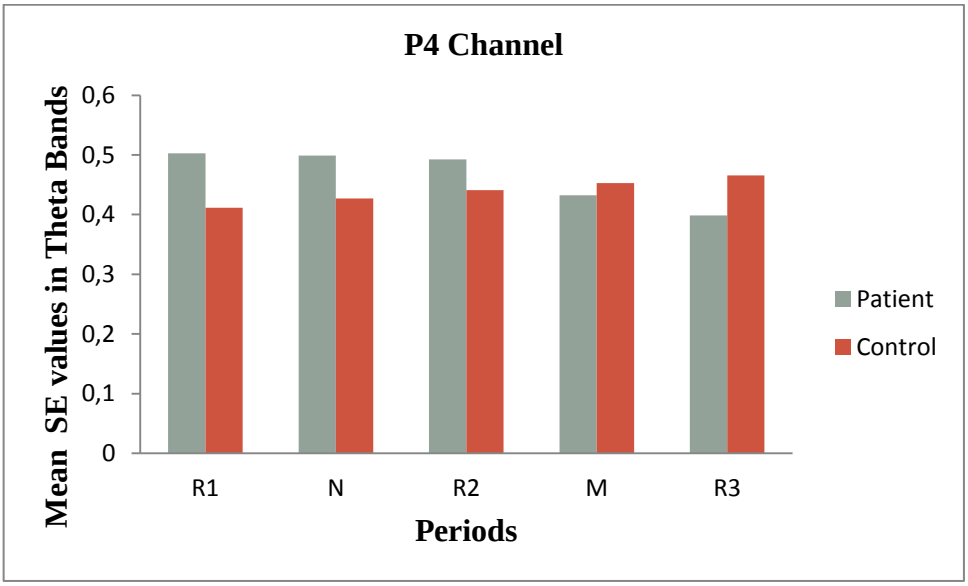
Figure 4.23 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE values of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing after R2 periods. The mean of SE values of groups is nearly same at R3 state.

Table 4.24 Comparison of SE and p values of Theta band during all measurement periods between patients and controls in P4 channel

Bands	Periods	Mean ±Std Deviation	Mean ± Std Deviation	p value
		Control	Patient	
Theta	R1	-0,411370±0,061208	-0,502600±0,169819	0,127
Theta	N	-0,427130±0,044452	-0,498920±0,168676	0,210
Theta	R2	-0,441220±0,051852	-0,492640±0,177354	0,390
Theta	M	-0,452850±0,071349	-0,432580±0,111976	0,635
Theta	R3	-0,465620±0,056132	-0,398690±0,091784	0,065*

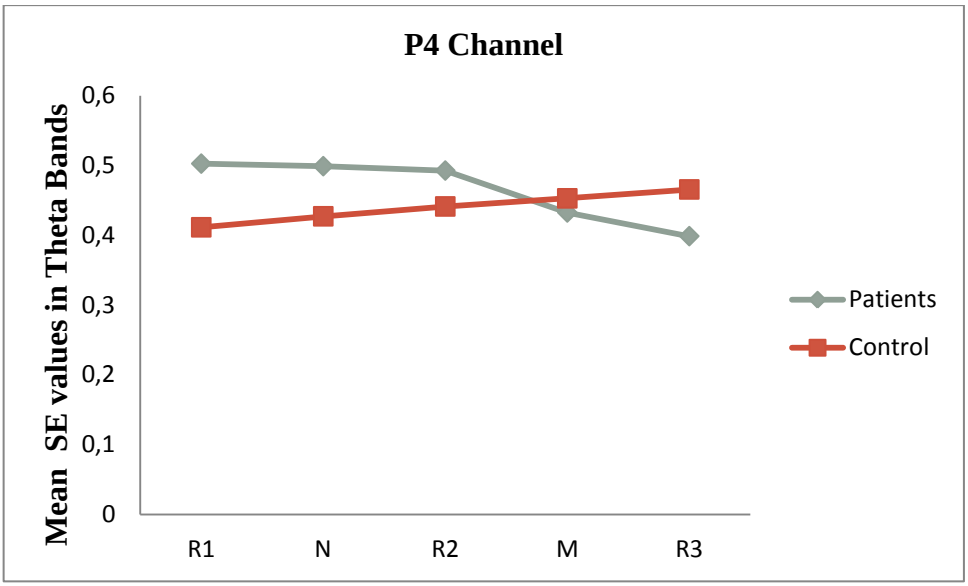
*p≤ 0, 05 is accepted for significant difference

Table 4.24 shows the significant difference is at R3 state.



(a)

Figure 4.24 (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of P4 channel in patient and control groups



(b)

Figure 4.24 (Continue) (a) shows that changes in the theta band with column graph and (b) shows that changes in the theta band with line graph of P4 channel in patient and control groups

Figure 4.24 shows that while red column and line graphs are indicating controls, grey column and line graphs show patients. The mean of SE of patients is higher than controls in R1, N, and R2 periods. The mean of SE values of patients is decreaing after R2 periods. The mean of SE values of groups are nearly same at M period.

Paired sample Student's t-test was applied to data, for analyzing the difference between sequential periods in patients and controls. Significant difference was approved when p value is less than 0, 05. Statistical comparisons were performed within each channel. P values mean and standart deviation comparisons between periods of each channel of patients are shown in Table 4.25-36. p values, mean and standart deviation comparisons between periods of each channel of healty groups are shown in Table 4.37-48.

Table 4.25 Comparison of extracted features in F3 region EEG data (alpha and delta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,013030± 0,038582	0,313	Alpha band	R1-N	-0,012380± 0,039525	0,348
Delta band	N-R2	0,016830± 0,034465	0,157	Alpha band	N-R2	0,063330± 0,151112	0,218
Delta band	R2- M	-0,089380± 0,223689	0,238	Alpha band	R2- M	-0,111100± 0,289580	0,256
Delta band	M-R3	-0,020340± 0,102870	0,547	Alpha band	M-R3	-0,023120± 0,100356	0,485
Delta band	R1-R2	0,029860± 0,050110	0,092	Alpha band	R1-R2	0,050950± 0,153031	0,320
Delta band	R1-M	-0,059520± 0,187985	0,343	Alpha band	R1-M	-0,060150± 0,288037	0,526
Delta band	R1-R3	-0,079860± 0,206467	0,252	Alpha band	R1-R3	-0,083270± 0,265458	0,347
Delta band	N-M	-0,072550± 0,225997	0,337	Alpha band	N-M	-0,047770± 0,284038	0,608
Delta band	N-R3	-0,092890± 0,240096	0,252	Alpha band	N-R3	-0,070890± 0,270752	0,429
Delta band	R2-R3	-0,109720± 0,232798	0,170	Alpha band	R2-R3	-0,134220± 0,279537	0,163

Table 4.25 shows that there is no significant difference for alpha and delta bands between sequential periods for patients in F3 channel.

Table 4.26 Comparison of extracted features in F3 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,006810± 0,048355	0,667	Beta band	R1-N	0,006300± 0,096816	0,842
Theta band	N-R2	0,006050± 0,054645	0,734	Beta band	N-R2	0,027520± 0,096133	0,389
Theta band	R2- M	-0,101500± 0,269893	0,265	Beta band	R2- M	-0,083000± 0,232871	0,289
Theta band	M-R3	0,001170± 0,063808	0,955	Beta band	M-R3	0,006380± 0,061647	0,751
Theta band	R1-R2	-0,000760± 0,077302	0,976	Beta band	R1-R2	0,033820± 0,106906	0,343
Theta band	R1-M	-0,102260± 0,261854	0,248	Beta band	R1-M	-0,049180± 0,237555	0,529
Theta band	R1-R3	-0,101090± 0,265422	0,259	Beta band	R1-R3	-0,042800± 0,257752	0,612
Theta band	N-M	-0,095450± 0,267222	0,288	Beta band	N-M	-0,055480± 0,219666	0,445
Theta band	N-R3	-0,094280± 0,266307	0,292	Beta band	N-R3	-0,049100± 0,230819	0,518
Theta band	R2-R3	-0,100330± 0,267261	0,266	Beta band	R2-R3	-0,076620± 0,258398	0,373

Table 4.26 shows that there is no significant difference for theta and beta bands between sequential periods for patients at F3 channel .

Table 4.27 Comparison of extracted features in F4 region EEG data (delta and alpha band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,041620± 0,093215	0,192	Alpha band	R1-N	-0,004060± 0,131567	0,924
Delta band	N-R2	0,007620± 0,047913	0,627	Alpha band	N-R2	0,030460± 0,139595	0,508
Delta band	R2- M	-0,070580± 0,209480	0,314	Alpha band	R2- M	-0,121850± 0,260036	0,173
Delta band	M-R3	0,059780± 0,233062	0,438	Alpha band	M-R3	0,062780± 0,285780	0,505
Delta band	R1-R2	0,049240± 0,112850	0,201	Alpha band	R1-R2	0,026400± 0,133948	0,549
Delta band	R1-M	-0,021340± 0,116876	0,578	Alpha band	R1-M	-0,095450± 0,258982	0,274
Delta band	R1-R3	0,038440± 0,125029	0,356	Alpha band	R1-R3	-0,032670± 0,092616	0,294
Delta band	N-M	-0,062960± 0,204666	0,356	Alpha band	N-M	-0,091390± 0,283035	0,334
Delta band	N-R3	-0,003180± 0,047400	0,837	Alpha band	N-R3	-0,028610± 0,119967	0,470
Delta band	R2-R3	-0,010800± 0,055422	0,553	Alpha band	R2-R3	-0,059070± 0,156323	0,263

Table 4.27 shows that there is no significant difference for delta and alpha bands between sequential periods for patients at F4 channel.

Table 4.28 Comparison of extracted features in F4 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,012270± 0,055430	0,502	Beta band	R1-N	-0,004680± 0,146897	0,922
Theta band	N-R2	0,029030± 0,104524	0,403	Beta band	N-R2	0,012200± 0,072089	0,606
Theta band	R2- M	-0,111820± 0,272933	0,227	Beta band	R2- M	-0,081950± 0,215920	0,261
Theta band	M-R3	0,071190± 0,275233	0,435	Beta band	M-R3	0,077190± 0,235973	0,328
Theta band	R1-R2	0,016760± 0,114605	0,655	Beta band	R1-R2	0,007520± 0,118908	0,846
Theta band	R1-M	-0,095060± 0,261120	0,279	Beta band	R1-M	-0,074430± 0,256676	0,383
Theta band	R1-R3	-0,023870± 0,067625	0,293	Beta band	R1-R3	0,002760± 0,220911	0,969
Theta band	N-M	-0,082790± 0,264335	0,348	Beta band	N-M	-0,069750± 0,211047	0,323
Theta band	N-R3	-0,011600± 0,047243	0,457	Beta band	N-R3	0,007440± 0,111405	0,837
Theta band	R2-R3	-0,040630± 0,089355	0,184	Beta band	R2-R3	-0,004760± 0,161103	0,928

Table 4.28 shows that there are no significant differences for theta and beta bands between sequential periods for patients at F4 channel.

Table 4.29 Comparison of extracted features in C3 region EEG data (alpha and delta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,008040± 0,118034	0,834	Alpha band	R1-N	0,010330± 0,090817	0,727
Delta band	N-R2	0,040120± 0,109486	0,276	Alpha band	N-R2	0,048440± 0,162760	0,371
Delta band	R2- M	-0,118270± 0,240494	0,154	Alpha band	R2- M	-0,132600± 0,290475	0,183
Delta band	M-R3	0,008090± 0,021554	0,266	Alpha band	M-R3	-0,016030± 0,084896	0,565
Delta band	R1-R2	0,048160± 0,092732	0,135	Alpha band	R1-R2	0,058770± 0,116231	0,144
Delta band	R1-M	-0,070110± 0,154718	0,186	Alpha band	R1-M	-0,073830± 0,269081	0,408
Delta band	R1-R3	-0,062020± 0,160963	0,254	Alpha band	R1-R3	-0,089860± 0,258812	0,301
Delta band	N-M	-0,078150± 0,198937	0,246	Alpha band	N-M	-0,084160± 0,257146	0,328
Delta band	N-R3	-0,070060± 0,202718	0,303	Alpha band	N-R3	-0,100190± 0,258223	0,251
Delta band	R2-R3	-0,110180± 0,244723	0,188	Alpha band	R2-R3	-0,148630± 0,283851	0,132

Table 4.29 shows that there is no significant difference for delta and alpha bands between sequential periods for patients at C3 channel.

Table 4.30 Comparison of extracted features in C3 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	0,002100± 0,060805	0,915	Beta band	R1-N	-0,031170± 0,064259	0,159
Theta band	N-R2	0,013900± 0,082661	0,608	Beta band	N-R2	0,023100± 0,117403	0,549
Theta band	R2- M	-0,057920± 0,315509	0,576	Beta band	R2- M	-0,066730± 0,248158	0,417
Theta band	M-R3	-0,003080± 0,045104	0,834	Beta band	M-R3	0,007500± 0,085963	0,789
Theta band	R1-R2	0,016000± 0,098974	0,622	Beta band	R1-R2	-0,008070± 0,091532	0,787
Theta band	R1-M	-0,04192± 0,307521	0,677	Beta band	R1-M	-0,074800± 0,247440	0,364
Theta band	R1-R3	-0,045000± 0,302210	0,649	Beta band	R1-R3	-0,067300± 0,272199	0,454
Theta band	N-M	-0,044020± 0,306194	0,660	Beta band	N-M	-0,043630± 0,262110	0,611
Theta band	N-R3	-0,047100± 0,302737	0,635	Beta band	N-R3	-0,036130± 0,270832	0.683
Theta band	R2-R3	-0,061000± 0,303507	0,541	Beta band	R2-R3	-0,059230± 0,277111	0,516

Table 4.30 shows that there is no significant difference for theta and beta bands between sequential periods for patients at C3 channel.

Table 4.31 Comparison of extracted features in C4 region EEG data (alpha and delta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,033950± 0,116672	0,381	Alpha band	R1-N	-0,005390± 0,189877	0,930
Delta band	N-R2	-0,003020± 0,007530	0,237	Alpha band	N-R2	-0,037910± 0,103371	0,276
Delta band	R2- M	-0,064210± 0,203275	0,344	Alpha band	R2- M	-0,040070± 0,277492	0,659
Delta band	M-R3	0,000060± 0,003781	0,961	Alpha band	M-R3	-0,029650± 0,061101	0,159
Delta band	R1-R2	0,030930± 0,112901	0,409	Alpha band	R1-R2	-0,043300± 0,109489	0,243
Delta band	R1-M	-0,033280± 0,091452	0,279	Alpha band	R1-M	-0,083370± 0,275390	0,363
Delta band	R1-R3	-0,033220± 0,091796	0,282	Alpha band	R1-R3	-0,113020± 0,296958	0,259
Delta band	N-M	-0,067230± 0,207147	0,345	Alpha band	N-M	-0,077980± 0,278126	0,398
Delta band	N-R3	-0,067170± 0,207200	0,332	Alpha band	N-R3	-0,107630± 0,260359	0,224
Delta band	R2-R3	-0,064150± 0,203343	0,332	Alpha band	R2-R3	-0,069720± 0,279709	0,451

Table 4.31 shows that there is no significant difference for delta and alpha bands between sequential periods for patients at C4 channel.

Table 4.32 Comparison of extracted features in C4 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	0,061870± 0,138907	0,193	Beta band	R1-N	0,033880± 0,112180	0,364
Theta band	N-R2	-0,003020± 0,007530	0,237	Beta band	N-R2	-0,008550± 0,086484	0,762
Theta band	R2- M	-0,064210± 0,203275	0,344	Beta band	R2- M	-0,068400± 0,215808	0,342
Theta band	M-R3	0,000060± 0,003781	0,961	Beta band	M-R3	0,019770± 0,208532	0,771
Theta band	R1-R2	0,058850± 0,136443	0,206	Beta band	R1-R2	0,025330± 0,099937	0,443
Theta band	R1-M	-0,005360± 0,135705	0,903	Beta band	R1-M	-0,043070± 0,214521	0,541
Theta band	R1-R3	-0,005300± 0,135672	0,904	Beta band	R1-R3	-0,023300± 0,332529	0,830
Theta band	N-M	-0,067230± 0,207147	0,332	Beta band	N-M	-0,076950± 0,214632	0,286
Theta band	N-R3	-0,067170± 0,207200	0,332	Beta band	N-R3	-0,057180± 0,276382	0,529
Theta band	R2-R3	-0,064150± 0,203343	0,345	Beta band	R2-R3	-0,048630± 0,325684	0,648

Table 4.32 shows that there is no significant difference for theta and beta bands between sequential periods for patients at C4 channel.

Table 4.33 Comparison of extracted features in P3 region EEG data (alpha and delta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,007491± 0,027501	0,388	Alpha band	R1-N	0,027010± 0,077341	0,298
Delta band	N-R2	-0,001718± 0,014659	0,706	Alpha band	N-R2	-0,006700± 0,102116	0,840
Delta band	R2- M	-0,064127± 0,213755	0,343	Alpha band	R2- M	-0,095220± 0,264232	0,284
Delta band	M-R3	-0,002409± 0,004977	0,139	Alpha band	M-R3	0,008400± 0,034554	0,462
Delta band	R1-R2	0,005773± 0,035476	0,601	Alpha band	R1-R2	0,020310± 0,070477	0,386
Delta band	R1-M	-0,058355± 0,187663	0,327	Alpha band	R1-M	-0,074910± 0,269634	0,402
Delta band	R1-R3	-0,060764± 0,186627	0,306	Alpha band	R1-R3	-0,066510± 0,273833	0,462
Delta band	N-M	-0,065845± 0,201638	0,304	Alpha band	N-M	-0,101920± 0,265785	0,256
Delta band	N-R3	-0,068255± 0,200835	0,286	Alpha band	N-R3	-0,093520± 0,273546	0,308
Delta band	R2-R3	-0,066536± 0,212958	0,324	Alpha band	R2-R3	-0,086820± 0,266281	0,329

Table 4.33 shows that there is no significant difference for delta and alpha bands between sequential periods for patients at P3 channel.

Table 4.34 Comparison of extracted features in P3 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,005580± 0,056438	0,762	Beta band	R1-N	0,081255± 0,162020	0,112
Theta band	N-R2	0,014560± 0,044573	0,329	Beta band	N-R2	-0,064545± 0,164466	0,222
Theta band	R2- M	-0,083060± 0,272004	0,359	Beta band	R2- M	-0,078555± 0,227665	0,279
Theta band	M-R3	-0,095400± 0,298551	0,339	Beta band	M-R3	-0,012591± 0,044618	0,371
Theta band	R1-R2	0,008980± 0,070587	0,697	Beta band	R1-R2	0,016709± 0,079576	0,502
Theta band	R1-M	-0,074080± 0,277551	0,421	Beta band	R1-M	-0,061845± 0,223137	0,380
Theta band	R1-R3	-0,169480± 0,351230	0,161	Beta band	R1-R3	-0,074436± 0,228301	0,305
Theta band	N-M	-0,068500± 0,272445	0,447	Beta band	N-M	-0,143100± 0,214735	0,052*
Theta band	N-R3	-0,163900± 0,360224	0,184	Beta band	N-R3	-0,155691± 0,217629	0,039*
Theta band	R2-R3	-0,178460± 0,355186	0,147	Beta band	R2-R3	-0,091145± 0,233295	0,224

Table 4.34 shows that there is significant differences for theta and beta bands between N-M (p=0,052) and N-R3 (p=0,039) periods at P3 channel in patients.

Table 4.35 Comparison of extracted features in P4 region EEG data (theta and beta band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,003680± 0,065018	0,862	Beta band	R1-N	-0,013470± 0,056618	0,471
Theta band	N-R	-0,006280± 0,044557	0,666	Beta band	N-R2	0,010790± 0,121399	0,785
Theta band	R2- M	-0,060060± 0,279291	0,514	Beta band	R2- M	-0,088460± 0,220919	0,237
Theta band	M-R3	-0,033890± 0,056026	0,088	Beta band	M-R3	0,031250± 0,159287	0,550
Theta band	R1-R2	-0,009960± 0,073288	0,677	Beta band	R1-R2	-0,002680± 0,103087	0,936
Theta band	R1-M	-0,070020± 0,270803	0,435	Beta band	R1-M	-0,091140± 0,206558	0,196
Theta band	R1-R3	-0,103910± 0,256600	0,239	Beta band	R1-R3	-0,059890± 0,249968	0,468
Theta band	N-M	-0,066340± 0,270981	0,459	Beta band	N-M	-0,077670± 0,211292	0,275
Theta band	N-R3	-0,100230± 0,256958	0,249	Beta band	N-R3	-0,046420± 0,258765	0,584
Theta band	R2-R3	-0,093950± 0,263811	0,289	Beta band	R2-R3	0,057210± 0,308196	0,572

Table 4.35 shows that there is no significant difference for theta and beta bands between periods for patients at P4 channel.

Table 4.36 Comparison of extracted features in P4 region EEG data (delta and alpha band) between sequential periods in patients

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,038230± 0,122567	0,862	Alpha band	R1-N	-0,013470± 0,056618	0,350
Delta band	N-R	0,003020± 0,014608	0,666	Alpha band	N-R2	0,010790± 0,121399	0,530
Delta band	R2- M	-0,070750± 0,222389	0,514	Alpha band	R2- M	-0,088460± 0,220919	0,341
Delta band	M-R3	0,001340± 0,010207	0,088	Alpha band	M-R3	0,031250± 0,159287	0,688
Delta band	R1-R2	0,041250± 0,131550	0,677	Alpha band	R1-R2	-0,002680± 0,103087	0,347
Delta band	R1-M	-0,029500± 0,092817	0,435	Alpha band	R1-M	-0,091140± 0,206558	0,337
Delta band	R1-R3	-0,028160± 0,092817	0,239	Alpha band	R1-R3	-0,059890± 0,249968	0,362
Delta band	N-M	-0,067730± 0,213438	0,459	Alpha band	N-M	-0,077670± 0,211292	0,342
Delta band	N-R3	-0,066390± 0,213761	0,249	Alpha band	N-R3	-0,046420± 0,258765	0,352
Delta band	R2-R3	-0,069410± 0,222262	0,289	Alpha band	R2-R3	0,057210± 0,308196	0,349

Table 4.36 shows that there are no significant differences for alpha and delta bands between periods for patients at P4 channel.

Table 4.37 Comparison of extracted features in F3 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	-0,004430± 0,027402	0,621	Alpha band	R1-N	0,057820± 0,194407	0,372
Delta band	N-R2	0,006860± 0,009891	0,056*	Alpha band	N-R2	-0,088320± 0,181936	0,159
Delta band	R2- M	0,003550± 0,014981	0,473	Alpha band	R2- M	0,070440± 0,173832	0,232
Delta band	M-R3	-0,002140± 0,010526	0,536	Alpha band	M-R3	-0,060940± 0,186971	0,330
Delta band	R1-R2	0,002430± 0,034209	0,827	Alpha band	R1-R2	-0,030500± 0,035580	0,024*
Delta band	R1-M	0,005980± 0,031410	0,562	Alpha band	R1-M	0,039940± 0,186892	0,516
Delta band	R1-R3	0,003840± 0,031539	0,709	Alpha band	R1-R3	-0,021000± 0,054609	0,255
Delta band	N-M	0,010410± 0,015708	0,066*	Alpha band	N-M	-0,017880± 0,057988	0,355
Delta band	N-R3	0,008270± 0,017608	0,172	Alpha band	N-R3	-0,078820± 0,202269	0,249
Delta band	R2-R3	0,001410± 0,016340	0,791	Alpha band	R2-R3	0,009500± 0,049840	0,562

Table 4.37 shows that there is significant differences for delta band between N-R2 (p=0,056) and there is no significant differences for alpha band between periods at F3 channel in controls.

Table 4.38 Comparison of extracted features in F3 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,001170± 0,026184	0,891	Beta band	R1-N	-0,007640± 0,069392	0,736
Theta band	N-R2	0,031340± 0,054008	0,100	Beta band	N-R2	0,007690± 0,101109	0,815
Theta band	R2- M	0,016130± 0,071708	0,495	Beta band	R2- M	0,001450± 0,132070	0,973
Theta band	M-R3	-0,030640± 0,046978	0,069*	Beta band	M-R3	-0,020240± 0,071827	0,396
Theta band	R1-R2	0,030170± 0,061551	0,156	Beta band	R1-R2	0,000050± 0,067145	0,998
Theta band	R1-M	0,046300± 0,053538	0,023	Beta band	R1-M	0,001500± 0,104045	0,965
Theta band	R1-R3	0,015660± 0,042404	0,273	Beta band	R1-R3	-0,018740± 0,075249	0,451
Theta band	N-M	0,047470± 0,062781	0,040*	Beta band	N-M	0,009140± 0,054183	0,607
Theta band	N-R3	016830± 0,035000	0,163	Beta band	N-R3	-0,011100± 0,050615	0,505
Theta band	R2-R3	-0,014510± 0,052924	0,408	Beta band	R2-R3	-0,018790± 0,116011	0,621

Table 4.38 shows that there is significant differences for theta band between M-R3 (p=0,069) and there is no significant differences for beta band between periods at F3 channel in controls.

Table 4.39 Comparison of extracted features in F4 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,009920± 0,022578	0,198	Alpha band	R1-N	0,016880± 0,090185	0,568
Delta band	N-R2	0,002460± 0,033542	0,822	Alpha band	N-R2	-0,018170± 0,086438	0,523
Delta band	R2- M	0,021470± 0,036622	0,097	Alpha band	R2- M	0,022610± 0,056875	0,240
Delta band	M-R3	-0,007760± 0,036679	0,520	Alpha band	M-R3	-0,007900± 0,098888	0,806
Delta band	R1-R2	0,012380± 0,037820	0,328	Alpha band	R1-R2	-0,001290± 0,025719	0,877
Delta band	R1-M	0,033850± 0,060878	0,113	Alpha band	R1-M	0,021320± 0,057511	0,271
Delta band	R1-R3	0,026090± 0,056152	0,176	Alpha band	R1-R3	0,013420± 0,070645	0,563
Delta band	N-M	0,023930± 0,059368	0,234	Alpha band	N-M	0,004440± 0,051039	0,789
Delta band	N-R3	0,016170± 0,046709	0,302	Alpha band	N-R3	-0,003460± 0,130179	0,935
Delta band	R2-R3	0,013710± 0,025748	0,127	Alpha band	R2-R3	0,014710± 0,070621	0,527

Table 4.39 shows that there is no significant difference for delta and alpha bands between periods at F4 channel in controls.

Table 4.40 Comparison of extracted features in F4 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,005090± 0,033504	0,642	Beta band	R1-N	-0,005180± 0,022055	0,447
Theta band	N-R2	0,001440± 0,025055	0,860	Beta band	N-R2	-0,00901± 0,020047	0,189
Theta band	R2- M	0,027170± 0,047930	0,107	Beta band	R2- M	0,023660± 0,053548	0,196
Theta band	M-R3	-0,003950± 0,038802	0,755	Beta band	M-R3	-0,011100± 0,051259	0,511
Theta band	R1-R2	-0,003650± 0,027687	0,687	Beta band	R1-R2	-0,014190± 0,023931	0,094
Theta band	R1-M	0,023520± 0,050691	0,176	Beta band	R1-M	0,009470± 0,052442	0,582
Theta band	R1-R3	0,019570± 0,031288	0,079	Beta band	R1-R3	-0,001630± 0,035104	0,886
Theta band	N-M	0,028610± 0,050053	0,104	Beta band	N-M	0,014650± 0,038993	0,265
Theta band	N-R3	0,024660± 0,022758	0,008*	Beta band	N-R3	0,003550± 0,027753	0,695
Theta band	R2-R3	0,023220± 0,025485	0,018*	Beta band	R2-R3	0,012560± 0,028408	0,196

Table 4.40 shows that there is no significant difference for theta and beta bands between periods at F4 channel in controls.

Table 4.41 Comparison of extracted features in C3 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,016750± 0,071114	0,475	Alpha band	R1-N	0,030950± 0,063465	0,157
Delta band	N-R2	-0,018540± 0,049505	0,267	Alpha band	N-R2	-0,019050± 0,096923	0,550
Delta band	R2- M	0,010860± 0,039641	0,409	Alpha band	R2- M	0,010690± 0,067237	0,627
Delta band	M-R3	0,000530± 0,026526	0,951	Alpha band	M-R3	-0,021660± 0,084366	0,438
Delta band	R1-R2	-0,001790± 0,051809	0,915	Alpha band	R1-R2	0,011900± 0,040972	0,382
Delta band	R1-M	0,009070± 0,058693	0,637	Alpha band	R1-M	0,022590± 0,033784	0,064*
Delta band	R1-R3	0,009600± 0,051323	0,569	Alpha band	R1-R3	0,000930± 0,073492	0,969
Delta band	N-M	-0,007680± 0,018989	0,233	Alpha band	N-M	-0,008360± 0,040809	0,533
Delta band	N-R3	-0,007150± 0,036682	0,553	Alpha band	N-R3	-0,030020± 0,107744	0,401
Delta band	R2-R3	0,011390± 0,026362	0,205	Alpha band	R2-R3	-0,010970± 0,078808	0,670

Table 4.41 shows that there is significant differences for alpha band between R1-M period and there is no differences for delta bands between periods at C3 channel in controls.

Table 4.42 Comparison of extracted features in C4 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean±Std. Dev.	p Value	Bands	Periods	Mean Std. Dev.	p Value
Theta band	R1-N	-0,005340± 0,019823	0,416	Beta band	R1-N	-0,001420± 0,042054	0,917
Theta band	N-R2	0,011760± 0,033803	0,300	Beta band	N-R2	-0,017510± 0,030600	0,104
Theta band	R2- M	0,018290± 0,040520	0,187	Beta band	R2- M	0,012980± 0,026489	0,156
Theta band	M-R3	-0,008930± 0,042253	0,521	Beta band	M-R3	0,003390± 0,043627	0,811
Theta band	R1-R2	0,006420± 0,033006	0,554	Beta band	R1-R2	-0,018930± 0,031834	0,093
Theta band	R1-M	0,024710± 0,056874	0,203	Beta band	R1-M	-0,005950± 0,046945	0,698
Theta band	R1-R3	0,015780± 0,038159	0,223	Beta band	R1-R3	-0,002560± 0,041811	0,851
Theta band	N-M	0,030050± 0,055146	0,119	Beta band	N-M	-0,004530± 0,025102	0,582
Theta band	N-R3	0,021120± 0,035767	0,095	Beta band	N-R3	-0,001140± 0,035392	0,921
Theta band	R2-R3	0,009360± 0,030776	0,361	Beta band	R2-R3	0,016370± 0,031466	0,134

Table 4.42 shows that there is no significant difference for theta and beta bands between periods at C4 channel in controls.

Table 4.43 Comparison of extracted features in C4 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,008530± 0,038098	0,497	Alpha band	R1-N	0,021540± 0,031338	0,058*
Delta band	N-R2	-0,011810± 0,016906	0,455	Alpha band	N-R2	0,010570± 0,048329	0,507
Delta band	R2- M	0,006380± 0,011793	0,121	Alpha band	R2- M	0,006100± 0,065187	0,707
Delta band	M-R3	0,000350± 0,019874	0,957	Alpha band	M-R3	0,004620± 0,061661	0,818
Delta band	R1-R2	-0,003280± 0,029099	0,730	Alpha band	R1-R2	0,032110± 0,029854	0,008*
Delta band	R1-M	0,003100± 0,033654	0,777	Alpha band	R1-M	0,038210± 0,050051	0,039*
Delta band	R1-R3	0,003450± 0,022841	0,644	Alpha band	R1-R3	0,042830± 0,058499	0,046*
Delta band	N-M	-0,005430± 0,007467	0,047	Alpha band	N-M	0,016670± 0,055073	0,363
Delta band	N-R3	-0,005080± 0,024490	0,528	Alpha band	N-R3	0,021290± 0,071674	0,372
Delta band	R2-R3	0,006730± 0,021250	0,343	Alpha band	R2-R3	0,010720± 0,061997	0,598

Table 4.43 shows that there is a significant difference for alpha band. There is a higher significant difference than other bands and there is no significant differences for delta, bands between periods at C4 channel in controls.

Table 4.44 Comparison of extracted features in C4 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	-0,000340± 0,031542	0,974	Beta band	R1-N	0,000140± 0,010362	0,967
Theta band	N-R2	0,015210± 0,056401	0,416	Beta band	N-R2	-0,008440± 0,015689	0,123
Theta band	R2- M	0,004300± 0,053319	0,804	Beta band	R2- M	0,027990± 0,060998	0,181
Theta band	M-R3	0,008320± 0,058099	0,661	Beta band	M-R3	-0,018140± 0,069467	0,430
Theta band	R1-R2	0,014870± 0,048549	0,358	Beta band	R1-R2	-0,008300± 0,018259	0,184
Theta band	R1-M	0,019170± 0,052142	0,275	Beta band	R1-M	0,019690± 0,053548	0,275
Theta band	R1-R3	0,027490± 0,056167	0,156	Beta band	R1-R3	0,001550± 0,030318	0,875
Theta band	N-M	0,019510± 0,058334	0,318	Beta band	N-M	0,019550± 0,050748	0,254
Theta band	N-R3	0,027830± 0,061782	0,188	Beta band	N-R3	0,001410± 0,033478	0,897
Theta band	R2-R3	0,012620± 0,033841	0,269	Beta band	R2-R3	0,009850± 0,029441	0,318

Table 4.44 shows that there is no significant difference for theta and beta bands between periods at C4 channel in controls.

Table 4.45 Comparison of extracted features in P3 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,031040± 0,030794	0,011*	Alpha band	R1-N	0,030910± 0,039273	0,034*
Delta band	N-R2	-0,013490± 0,024960	0,122	Alpha band	N-R2	-0,005450± 0,069966	0,811
Delta band	R2- M	0,009850± 0,041383	0,471	Alpha band	R2- M	0,007750± 0,067160	0,724
Delta band	M-R3	-0,006430± 0,036438	0,590	Alpha band	M-R3	-0,005280± 0,059265	0,785
Delta band	R1-R2	0,017550± 0,038754	0,186	Alpha band	R1-R2	0,025460± 0,045319	0,109
Delta band	R1-M	0,027400± 0,036695	0,043*	Alpha band	R1-M	0,033210± 0,041331	0,032*
Delta band	R1-R3	0,020970± 0,048492	0,205	Alpha band	R1-R3	0,027930± 0,048474	0,102
Delta band	N-M	-0,003640± 0,035687	0,754	Alpha band	N-M	0,002300± 0,034324	0,837
Delta band	N-R3	-0,010070± 0,054450	0,573	Alpha band	N-R3	-0,002980± 0,068296	0,893
Delta band	R2-R3	0,003420± 0,053762	0,845	Alpha band	R2-R3	0,002470± 0,032322	0,814

Table 4.45 shows that there is significant differences for delta band between R1-N periods and there is significant differences for alpha bands between R1-N and R1-M periods at P3 channel in controls.

Table 4.46 Comparison of extracted features in P3 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	0,005810± 0,054608	0,744	Beta band	R1-N	-0,005240± 0,014423	0,280
Theta band	N-R2	0,008120± 0,056036	0,658	Beta band	N-R2	-0,003420± 0,005699	0,090
Theta band	R2- M	0,034260± 0,049266	0,055*	Beta band	R2- M	0,001330± 0,003606	0,274
Theta band	M-R3	-0,019850± 0,062337	0,340	Beta band	M-R3	0,004230± 0,009084	0,175
Theta band	R1-R2	0,013930± 0,053498	0,432	Beta band	R1-R2	-0,008660± 0,013174	0,067*
Theta band	R1-M	0,048190± 0,071121	0,061*	Beta band	R1-M	-0,007330± 0,011925	0,084*
Theta band	R1-R3	0,028340± 0,048426	0,097	Beta band	R1-R3	-0,003100± 0,014981	0,529
Theta band	N-M	0,042380± 0,075437	0,109	Beta band	N-M	-0,002090± 0,005116	0,229
Theta band	N-R3	0,022530± 0,057129	0,244	Beta band	N-R3	0,002140± 0,010771	0,545
Theta band	R2-R3	0,014410± 0,041517	0,301	Beta band	R2-R3	0,005560± 0,008023	0,056*

Table 4.46 shows that there is significant differences for theta band between R2-M, R1-M periods and there is significant differences for beta bands between R1-R2, R1-R2 and R2-R3 periods at P3 channel in controls.

Table 4.47 Comparison of extracted features in P4 region EEG data (alpha and delta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Delta band	R1-N	0,008760± 0,016542	0,128	Alpha band	R1-N	0,030330± 0,072170	0,217
Delta band	N-R2	-0,006920± 0,022092	0,348	Alpha band	N-R2	-0,014510± 0,069988	0,528
Delta band	R2- M	0,013070± 0,048113	0,413	Alpha band	R2- M	0,051070± 0,073801	0,056*
Delta band	M-R3	0,004720± 0,052481	0,783	Alpha band	M-R3	-0,020690± 0,095479	0,510
Delta band	R1-R2	0,001840± 0,011099	0,613	Alpha band	R1-R2	0,015820± 0,077678	0,536
Delta band	R1-M	0,014910± 0,047972	0,351	Alpha band	R1-M	0,066890± 0,084134	0,033*
Delta band	R1-R3	0,019630± 0,035855	0,117	Alpha band	R1-R3	0,046200± 0,093900	0,154
Delta band	N-M	0,006150± 0,047145	0,690	Alpha band	N-M	0,036560± 0,043673	0,027 *
Delta band	N-R3	0,010870± 0,045103	0,465	Alpha band	N-R3	0,015870± 0,089122	0,587
Delta band	R2-R3	0,017790± 0,040084	0,194	Alpha band	R2-R3	0,030380± 0,041981	0,048*

Table 4.47 shows that there is significant differences for alpha band between R2-M, R1-M, N-M and R3-R3 periods and there is no significant differences for delta bands periods at P4 channel in controls.

Table 4.48 Comparison of extracted features in P4 region EEG data (theta and beta band) between sequential periods in controls

Bands	Periods	Mean± Std. Dev.	p Value	Bands	Periods	Mean± Std. Dev.	p Value
Theta band	R1-N	0,015760± 0,033851	0,175	Beta band	R1-N	0,003590± 0,011315	0,342
Theta band	N-R2	0,014090± 0,030700	0,181	Beta band	N-R2	-0,007850± 0,011243	0,055*
Theta band	R2- M	0,011630± 0,051593	0,494	Beta band	R2- M	0,009330± 0,025881	0,284
Theta band	M-R3	0,012770± 0,046258	0,405	Beta band	M-R3	-0,004860± 0,039413	0,706
Theta band	R1-R2	0,029850± 0,036372	0,029*	Beta band	R1-R2	-0,004260± 0,011432	0,269
Theta band	R1-M	0,041480± 0,051591	0,032	Beta band	R1-M	0,005070± 0,023370	0,510
Theta band	R1-R3	0,054250± 0,064346	0,026*	Beta band	R1-R3	0,000210± 0,032875	0,984
Theta band	N-M	0,025720± 0,065589	0,246	Beta band	N-M	0,001480± 0,022499	0,840
Theta band	N-R3	0,038490± 0,057322	0,63	Beta band	N-R3	-0,003380± 0,025969	0,690
Theta band	R2-R3	0,024400± 0,044279	0,115	Beta band	R2-R3	0,004470± 0,025702	0,596

Table 4.48 shows that there is significant differences for theta band between R1-R2, R1-R3 periods and there is significant differences for beta bands between N-R2 periods at P4 channel in controls.

CHAPTER 5

DISCUSSION

In recent years, psychiatric disorders are increaing rapidly in our country and our world. One of the most dangerous disease is panic disorder and result of this increasing panic attacks.

Human brain can not be directly observed and scientists use indirect techniques because of not taking pieces from brains unlike other organs. Biochemical disorders of brain can be investigated in laboratory and imaging of brain structure gives information about biological structure in psychiatry. On the other hand bioelectrical functions are measured by EEG. Electrophysiological data and variables are expressed for differential diagnosis, monitoring, determining response of treatment in psychiatry. Psychiatric disorders effects waves of brain in the literature. In adults, young adults and childrens with psychological disorders such as panic disorder, the treatment with measuring of brain functions is wanted goal.

Generally, scientists investigate prolongation of brain wave by using auditory stimulus. In 2002, Tayfun Turan and his friends investigate event-related potential (ERP) changes in panic disorder. ERPs were recorded by using auditory ‘‘odd-ball two-tone discrimination task’’ method. It was found that there was significant prolongation in P3 latency in the PD [129]. Jijun Wang and his friend used to target to explain amplitude of negative 200 which is brain wave, reduced in PD patients using oddball paradigm in 2003 [130]. In previous studies, EEG signals were analyzed in time axis.

The aim of our study is to save the brain signals and determine how brain waves change using advanced engineering methods (for the signals obtained, using of wavelet transform and the dividing into EEG lower frequency to take the entropy), compare changes between the records and evaluate by giving main auditory stimuli (the sound of an ambulance and relaxing sound) to the panic disorder patients by electroencephalography (EEG). The record began with resting state (R1), after N period

again resting state started (R2) and after M period, last resting state (R3) began. During the recording patients and control groups sat up chair and they did not move. Their eyes closed during EEG data recording. Recorded EEG signals were analysed in MATLAB withusing discreate wavelet decomposition. It decomposed data to sub bands such as alpha, beta, delta, and theta. Independent sample t-test and paired sample t-test were used to explain significantly differences.

After signal processing, bands showed some differences between periods. When values of patients and controls data are evaluated, some changes are observed with using independent sample t-test program.

Especially beta bands in all periods show high significant differences that can be discriminate both groups from each other in all channels. Beta band is more significant in all channels. Beta band is high frequency band. Beta waves are connected with active brain and alert state of mind [131]. Alpha bands have no significant differences in channel without F3 and F4 channel. Delta and theta didn't show significant differences in F4, C3, C4, P3 channels.

Theta waves are associated with drowsiness of an individual. Frontal midline theta (named like this because of its localization on scalp) is seen during a large variety of tasks like mental calculation. According to a study, pleasant music increases frontal midline theta power [132]. In my study theta band has a significant difference in R1 state in F3 channel and in R3 period. We may say our relaxation music does not much related to drowsiness of individual and aren't very effective on theta band.

In most channel of patients, the mean of SE vlaues is increasing until M period. Noise increases mean of SE values of patients. After listening music, the mean of SE values are decreasing in patients. This is expected result. The music makes a relax to panic disorder patients. In generally, controls doesn't effect by relaxing music.

Limitations of the this study are that the number of subjects is not so much for significant differences statistically. In another study, the procedure can be changed. Ten minutes procedure with eyes closed, may felt long to subjects. Time can be shortened. Procedure can be only a resting and a noise periods. During recording of EEG data, subjects were under stress and they sweat. The room where the patient may be large and spacious. Different types of sound can be tested at different times. Because it was

difficult to convince the subjects to procedure, there was not much subject. More subjects can be used for more accurate statistic measurements.

As a conclusion, the distinctive features between panic disorder patients and controls based on EEG signals with the help of advanced engineering methods were investigated. They are thought that they are useful attributes. Design of expert systems are considered for objective distinguished features in psychiatry and relaxing music may be listened to the patients with panic disorder to be relax.

REFERENCES

[1] Brainproduct details, http://www.brainproducts.com/productdetails.php?id=15, Access time: 15.04.2015

[2] Gorman JM, Liebowitz MR, Fyer AJ, Stein J. A neuroanatomical hypothesis for panic disorder. The American journal of psychiatry 1989.

[3] Davis M, Whalen PJ. The amygdala: vigilance and emotion. Molecular psychiatry 2001;6:13-34.

[4] Arıkan PDK. TÜRKİYE'DE GENEL TIP VE PSİKİYATRİ.

[5] Erginöz E. HALK SAĞLIĞI ve MENTAL HASTALIKLAR.

[6] Ertan PDT. PSİKİYATRİK BOZUKLUKLARIN EPİDEMİYOLOJİSİ.

[7] Bakanlığı S, Osman BOPDM. OBSESİF KOMPULSİF BOZUKLUK VE PANİK BOZUKLUĞU HASTALARINDAKİ CİNSEL İŞLEV BOZUKLUKLARININ KARŞILAŞTIRILMASI.

[8] Sadock BJ, Kaplan HI, Sadock VA. Kaplan & Sadock's synopsis of psychiatry: behavioral sciences/clinical psychiatry: Lippincott Williams & Wilkins; 2007.

[9] Karakula S. Koroner By-Pass Ameliyatı Geçiren Hastalarda Bilgilendirici Hemşirelik Yaklaşımının Ameliyat Öncesi ve Sonrası Anksiyete Düzeylerine Etkisinin İncelenmesi. Y Lisans Tezi, İzmir 1999.

[10] Öztürk MO, Uluşahin A. Ruh sağlığı ve bozuklukları: Nobel Tıp Kitapları; 2011.

[11] Gratacòs M, Nadal M, Martín-Santos Ro, Pujana MA, Gago J, Peral B, et al. A polymorphic genomic duplication on human chromosome 15 is a susceptibility factor for panic and phobic disorders. Cell 2001;106:367-79.

[12] Elmore KO, Schneider RK. Anxiety disorders in women. Women's Health in Primary Care 2001;4:691-8.

[13] Alonso J, Angermeyer MC, Bernert S, Bruffaerts R, Brugha T, Bryson H, et al. Prevalence of mental disorders in Europe: results from the European Study of the Epidemiology of Mental Disorders (ESEMeD) project. Acta Psychiatr Scand 2004;109:21-7.

[14] KOCABAŞOĞLU N. Anksiyete Bozukluklarına Acil Yaklaşım, Takip ve Tedavi. Türkiye Klinikleri Cerrahi Tıp Bilimleri Dergisi 2007;3:24-31.

[15] Arkonaç O. Açıklamalı Psikiyatri Sözlüğü. İstanbul: Nobel Tıp Kitabevleri Ltd Şti; 1999.

[16] Association AP. The Diagnostic and Statistical Manual of Mental Disorders: DSM 5: bookpointUS; 2013.

[17] Klein DF. Delineation of Two Drug-Responsive Anxiety Syndromes. Psychopharmacologia 1964;5:397-408.

[18] Roy-Byrne PP, Craske MG, Stein MB. Panic disorder. Lancet 2006;368:1023-32.

[19] Roy-Byrne PP, Craske MG, Stein MB. Panic disorder. The Lancet 2006;368:1023-32.

[20] Mattis SG, Ollendick TH. Panic in Children and Adolescents. Advances in clinical child psychology: Springer; 1997. p. 27-74.

[21] Gorman JM, Liebowitz MR, Fyer AJ, Stein J. A neuroanatomical hypothesis for panic disorder. The American journal of psychiatry 1989;146:148-61.

[22] Rifkin A, Siris SG. Panic disorder: response to sodium lactate and treatment with antidepressants. Progress in Neuro-Psychopharmacology and Biological Psychiatry 1985;9:33-8.

[23] TEZİ RSVHU, LEVENT DBA. PANİK BOZUKLUKTA YAŞAM KALİTESİ: 3 AYLIK İZLEM ÇALIŞMASI.

[24] Goddard AW, Charney DS. Toward an integrated neurobiology of panic disorder. Journal of Clinical Psychiatry 1997.

[25] Pollack M, Marzol P. Panic: course, complications and treatment of panic disorder. Journal of psychopharmacology (Oxford, England) 1999;14:S25-30.

[26] Tükel R. Anksiyete bozuklukları. Çizgi Tıp Yayınları Ankara 2000.

[27] Barlow DH. Anxiety and its disorders: The nature and treatment of anxiety and panic: Guilford press; 2004.

[28] Nardi AE, Freire RC, Zin WA. Panic disorder and control of breathing. Respir Physiol Neurobiol 2009;167:133-43.

[29] Association AP. Diagnostic And Statistical Manual Of Mental Disorders DSM-IV-TR Fourth Edition (Text Revision) Author: American Psychiatr. 2000.

[30] Pecknold JC, Luthe L. Sleep studies and neurochemical correlates in panic disorder and agoraphobia. Progress in Neuro-Psychopharmacology and Biological Psychiatry 1990;14:753-8.

[31] Kircanski K, Craske MG, Epstein AM, Wittchen HU. Subtypes of panic attacks: a critical review of the empirical literature. Depress Anxiety 2009;26:878-87.

[32] Agargün MY, Algün E, Şekeroğlu R, Kara H, Tarakçioğlu M. Low cholesterol level in patients with panic disorder: the association with major depression. J Affect Disord 1998;50:29-32.

[33] Freire RC, Valença AM, Nascimento I, Lopes FL, Mezzasalma MA, Zin WA, et al. Clinical features of respiratory and nocturnal panic disorder subtypes. Psychiatry Res 2007;152:287-91.

[34] Atalay N, Bayraktar E. Panik Atağı ve Panik Bozukluğu. Anksiyete Monografları Serisi 1995;6.

[35] Clin J. Psychiatry. 1998;59:24–8.

[36] J A. Psychiatry 2000:493–505.

[37] Whalen PJ, Phelps EA. The human amygdala: Guilford Press; 2009.

[38] Phelps EA, LeDoux JE. Contributions of the amygdala to emotion processing: from animal models to human behavior. Neuron 2005;48:175-87.

[39] Redmond Jr DE, Huang YH, Snyder DR, Maas JW. Behavioral effects of stimulation of the nucleus locus coeruleus in the stump-tailed monkey< i> Macaca arctoides</i>. Brain Res 1976;116:502-10.

[40] Charney DS, Woods SW, Nagy LM, Southwick SM, Krystal J, Heninger G. Noradrenergic function in panic disorder. Journal of Clinical Psychiatry 1990.

[41] Insel TR, Ninan PT, Aloi J, Jimerson DC, Skolnick P, Paul SM. A benzodiazepine receptor-mediated model of anxiety. Studies in nonhuman primates and clinical implications. Archives of general psychiatry 1984;41:741-50.

[42] Gray JA. The neuropsychology of anxiety. Issues in mental health nursing 1985;7:201-28.

[43] Reiman EM, Raichle ME, Butler FK, Herscovitch P, Robins E. A focal brain abnormality in panic disorder, a severe form of anxiety. Nature 1984;310:683-5.

[44] Memon MA. Panic Disorder.

[45] Vythilingam M, Anderson ER, Goddard A, Woods SW, Staib LH, Charney DS, et al. Temporal lobe volume in panic disorder—a quantitative magnetic resonance imaging study. Psychiatry Research: Neuroimaging 2000;99:75-82.

[46] Dratcu L. Panic, hyperventilation and perpetuation of anxiety. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2000;24:1069-89.

[47] Carr DB, Sheehan DV. Panic anxiety: a new biological model. Journal of Clinical Psychiatry 1984.

[48] Ebert MH, Birsöz S, Karaman T. Current psikiyatri: tanı ve tedavi: Güneş Kitabevi; 2003.

[49] Maron E, Hettema J, Shlik J. Advances in molecular genetics of panic disorder. Molecular psychiatry 2010;15:681-701.

[50] Pollack MH, Otto MW, Rosenbaum JF, Sachs GS, O'Neil C, Asher R, et al. Longitudinal course of panic disorder: findings from the Massachusetts General Hospital Naturalistic Study. The Journal of clinical psychiatry 1990;51 Suppl A:12-6.

[51] Noyes R, Jr., Clancy J, Hoenk PR, Slymen DJ. The prognosis of anxiety neurosis. Archives of general psychiatry 1980;37:173-8.

[52] Sadock BJ. Kaplan & Sadock's Comprehensive Textbook of Psychiatry (2 Volume Set): Lippincott Williams & Wilkins; 2000.

[53] Zun LS. Panic disorder: diagnosis and treatment in emergency medicine. Annals of emergency medicine 1997;30:92-6.

[54] Erdoğan S. Panik Bozukluğunun Nörobiyolojisi.

[55] Kocabaşoğlu N. Panik Bozukluğu, Agorafobi ve Diğer Komorbid Durumlar. Yeni Symposium Dergisi2002. p. 68-75.

[56] Taylor S, Asmundson GJ, Wald J. Psychopathology of panic disorder. Psychiatry 2007;6:188-92.

[57] Arch JJ, Craske MG. Addressing relapse in cognitive behavioral therapy for panic disorder: Methods for optimizing long-term treatment outcomes. Cogn Behav Pract 2011;18:306-15.

[58] Massion AO, Warshaw MG, Keller MB. Quality of life and psychiatric morbidity in panic disorder and generalized anxiety disorder. The American journal of psychiatry 1993.

[59] Katon W. Panic disorder: relationship to high medical utilization, unexplained physical symptoms, and medical costs. Journal of Clinical Psychiatry 1996.

[60] NOYES Jr R, Clancy J, Woodman C, Holt CS, Suelzer M, Christiansen J, et al. Environmental factors related to the outcome of panic disorder: a seven-year follow-up study. The Journal of nervous and mental disease 1993;181:529-38.

[61] Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: results from the National Comorbidity Survey. Archives of general psychiatry 1994;51:8-9.

[62] Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives of general psychiatry 2005;62:617-27.

[63] Kroenke K, Spitzer RL, Williams JB, Monahan PO, Löwe B. Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. Annals of internal medicine 2007;146:317-25.

[64] Greenberg PE, Sisitsky T, Kessler RC, Finkelstein SN, Berndt ER, Davidson JR, et al. The economic burden of anxiety disorders in the 1990s. Journal of Clinical Psychiatry 1999.

[65] Lee H, Hening W, Allen R, Kalaydjian A, Earley C, Eaton W, et al. Restless legs syndrome is associated with DSM-IV major depressive disorder and panic disorder in the community. The Journal of neuropsychiatry and clinical neurosciences 2008;20:101-5.

[66] Kaiya H, Sugaya N, Iwasa R, Tochigi M. Characteristics of fatigue in panic disorder patients. Psychiatry and clinical neurosciences 2008;62:234-7.

[67] Chen Y-H, Hu C-J, Lee H-C, Lin H-C. An increased risk of stroke among panic disorder patients: a 3-year follow-up study. Canadian journal of psychiatry Revue canadienne de psychiatrie 2010;55:43-9.

[68] Gomez-Caminero A, Blumentals WA, Russo LJ, Brown RR, Castilla-Puentes R. Does panic disorder increase the risk of coronary heart disease? A cohort study of a national managed care database. Psychosomatic medicine 2005;67:688-91.

[69] Fleet R, Lespérance F, Arsenault A, Grégoire J, Lavoie K, Laurin C, et al. Myocardial perfusion study of panic attacks in patients with coronary artery disease. The American journal of cardiology 2005;96:1064-8.

[70] Sullivan GM, Kent JM, Kleber M, Martinez JM, Yeragani VK, Gorman JM. Effects of hyperventilation on heart rate and QT variability in panic disorder pre-and post-treatment. Psychiatry research 2004;125:29-39.

[71] Schmidt NB, Lerew DR, Santiago H, Trakowski JH, Staab JP. Effects of heart-rate feedback on estimated cardiovascular fitness in patients with panic disorder. Depression and anxiety 2000;12:59-66.

[72] Chen Y-H, Lin H-C, Lee H-C. Pregnancy outcomes among women with panic disorder—Do panic attacks during pregnancy matter? Journal of affective disorders 2010;120:258-62.

[73] Katerndahl DA, Talamantes M. A comparison of persons with early-versus late-onset panic attacks. Journal of Clinical Psychiatry 2000;61:422-7.

[74] Hofmann SG, Smits JA. Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. The Journal of clinical psychiatry 2008;69:621.

[75] Doctor RM. Major results of a large-scale pretreatment survey of agoraphobics. Phobia: A comprehensive summary of modern treatments, Brunner/Mazel, New York 1982:203-14.

[76] Barsky AJ, Cleary PD, Coeytaux RR, Ruskin JN. Psychiatric disorders in medical outpatients complaining of palpitations. Journal of general internal medicine 1994;9:306-13.

[77] Fyer AJ, Sandberg D. Pharmacologic treatment of panic disorder. Review of psychiatry 1988;7:88-120.

[78] Association AP. Practice guidelines for the treatment of psychiatric disorders: compendium 2000;[psychiatric evaluation of adults; delirium; Alzheimer's disease and other dementias of late life; substance use disorders: alcohol, cocaine, opioids; nicotine dependence; schizophrenia; major depressive disorder ; bipolar disorder; panic disorder; eating disorders]: na; 2000.

[79] Taylor S. Understanding and treating panic disorder: Cognitive-behavioural approaches: John Wiley & Sons Ltd; 2000.

[80] Barlow DH. Clinical handbook of psychological disorders: A step-by-step treatment manual: Guilford publications; 2014.

[81] Beck AT, Dozois DJ. Cognitive therapy: current status and future directions. Annual review of medicine 2011;62:397-409.

[82] Deacon B, Abramowitz J. A pilot study of two-day cognitive-behavioral therapy for panic disorder. Behaviour Research and Therapy 2006;44:807-17.

[83] Yang Y, Kircher T, Straube B. The neural correlates of cognitive behavioral therapy: Recent progress in the investigation of patients with panic disorder. Behaviour Research and Therapy 2014.

[84] Koch EI, Gloster AT, Waller SA. Exposure treatments for panic disorder with and without agoraphobia. Handbook of exposure therapies 2007:221-45.

[85] Pitchot W, Ansseau M. Efficacy of quetiapine in treatment-resistant panic disorder: A case report. Asian journal of psychiatry 2012;5:204-5.

[86] Burrows GD, Judd FK, Norman TR. Long-term drug treatment of panic disorder. Journal of psychiatric research 1993;27:111-2

[87] Lydiard RB, Ballenger JC. Antidepressants in panic disorder and agoraphobia. Journal of affective disorders 1987;13:153-68.

[88] Ballenger JC. Efficacy of benzodiazepines in panic disorder and agoraphobia. Journal of psychiatric research 1990;24:15-25.

[89] Westenberg HG, Den Boer J. New findings in the treatment of panic disorder. Pharmacopsychiatry 1993.

[90] Pollack MH, Rosenbaum JF. Benzodiazepines in panic-related disorders. Journal of anxiety disorders 1988;2:95-107.

[91] Salzman C. Benzodiazepine treatment of panic and agoraphobic symptoms: use, dependence, toxicity, abuse. Journal of psychiatric research 1993;27:97-110.

[92] Berntson GG, Cacioppo JT. Heart rate variability: Stress and psychiatric conditions. Dynamic electrocardiography 2004:57-64.

[93] Yeragani VK, Tancer M, Uhde T. Heart rate and QT interval variability: abnormal alpha-2 adrenergic function in patients with panic disorder. Psychiatry research 2003;121:185-96.

[94] Pohl R, K Yeragani V. QT interval variability in panic disorder patients after isoproterenol infusions. The International Journal of Neuropsychopharmacology 2001;4:17-20.

[95] Weissman MM, Markowitz JS, Ouellette R, Greenwald S, Kahn JP. Panic disorder and cardiovascular/cerebrovascular problems: results from a community survey. The American journal of psychiatry 1990.

[96] Peker M. EEG sinyallerinden anomali tespiti

[97] Stein MB, Uhde TW. Infrequent occurrence of EEG abnormalities in panic disorder. The American journal of psychiatry 1989;146:517-20.

[98] Bystritsky A, Leuchter AF, Vapnik T. EEG abnormalities in nonmedicated panic disorder. The Journal of nervous and mental disease 1999;187:113-4.

[99] Turan M, Eşel E. Panik bozukluğun elektrofizyolojisi. Klinik Psikofarmakoloji 2002.

[100] Knott VJ, Bakish D, Lusk S, Barkely J, Perugini M. Quantitative EEG correlates of panic disorder. Psychiatry Research: Neuroimaging 1996;68:31-9.

[101] Dantendorfer K, Prayer D, Kramer J, Amering M, Baischer W, Berger P, et al. High frequency of EEG and MRI brain abnormalities in panic disorder. Psychiatry Research: Neuroimaging 1996;68:41-53.

[102] Edlund MJ, Swann AC, Clothier J. Patients with panic attacks and abnormal EEG results. The American journal of psychiatry 1987.

[103] Lepola U, Nousiainen U, Puranen M, Riekkinen P, Rimón R. EEG and CT findings in patients with panic disorder. Biological psychiatry 1990;28:721-7.

[104] Beauclair L, Fontaine R. Epileptiform abnormalities in panic disorder. Society of Biological Psychiatry, 41st Annual Convention and Scientific Program1986. p. 148.

[105] Yamasaki T, Ogata K, Maekawa T, Ijichi I, Katagiri M, Mitsudo T, et al. Rapid maturation of voice and linguistic processing systems in preschool children: a near-infrared spectroscopic study. Experimental neurology 2013;250:313-20.

[106] Di G-q, Li Z-g, Zhang B-j, Shi Y. Adjustment on subjective annoyance of low frequency noise by adding additional sound. Journal of Sound and Vibration 2011;330:5707-15.

[107] Smith S. EEG in the diagnosis, classification, and management of patients with epilepsy. Journal of Neurology, Neurosurgery & Psychiatry 2005;76:ii2-ii7.

[108] Chen G. Are electroencephalogram (EEG) signals pseudo-random number generators? Journal of Computational and Applied Mathematics 2014;268:1-4.

[109] Parvez MZ, Paul M. Epileptic Seizure Detection by Analyzing EEG Signals using Different Transformation Techniques. Neurocomputing 2014.

[110] Ray G. An algorithm to separate nonstationary part of a signal using mid-prediction filter. Signal Processing, IEEE Transactions on 1994;42:2276-9.

[111] Mammone N, La Foresta F, Morabito FC. Automatic artifact rejection from multichannel scalp EEG by wavelet ICA. Sensors Journal, IEEE 2012;12:533-42.

[112] Molla MKI, Islam R, Tanaka T, Rutkowski TM. Artifact suppression from EEG signals using data adaptive time domain filtering. Neurocomputing 2012;97:297-308.

[113] Kovach CK, Tsuchiya N, Kawasaki H, Oya H, Howard MA, 3rd, Adolphs R. Manifestation of ocular-muscle EMG contamination in human intracranial recordings. NeuroImage 2011;54:213-33.

[114] JASPER HH. The ten twenty electrode system of the international federation. Electroencephalography and clinical neurophysiology 1958;10:371-5.

[115] Seo Y, Kim S, Kisi O, Singh VP. Daily Water Level Forecasting Using Wavelet Decomposition and Artificial Intelligence Techniques. Journal of Hydrology 2014.

[116] Meng L, Xiang J, Wang Y, Jiang Y, Gao H. A hybrid fault diagnosis method using morphological filter–translation invariant wavelet and improved ensemble empirical mode decomposition. Mechanical Systems and Signal Processing 2015;50:101-15.

[117] NOURANI V, NEJAD FH, RAHIMI AY, BAGHANAM AH. Appraisal to utilization of wavelet denoising approach in ANN-based hydrological models.

[118] Hoekstra A, Janssen J. Linking bio-signals to transfer of knowledge: Towards mind-reading ecas. Capita Selecta 2006.

[119] Bhowmik P, Purkait P, Bhattacharya K. A novel wavelet transform aided neural network based transmission line fault analysis method. International Journal of Electrical Power & Energy Systems 2009;31:213-9.

[120] Misiti M. Wavelet Toolbox User's Guide, Version 1: For Use with MATLAB. Math Works; 1996.

[121] Kociołek M, Materka A, Strzelecki M, Szczypiński P. Discrete wavelet transform-derived features for digital image texture analysis. International Conference on Signals and Electronic Systems, Łódź-Poland2001. p. 99-104.

[122] Polikar R. The story of wavelets. Physics and modern topics in mechanical and electrical engineering 1999:192-7.

[123] Edwards T. Discrete wavelet transforms: Theory and implementation. Universidad de 1991.

[124] Bein B. Entropy. Best Practice & Research Clinical Anaesthesiology 2006;20:101-9.

[125] Gajic D, Djurovic Z, Di Gennaro S, Gustafsson F. Classification of EEG signals for detection of epileptic seizures based on wavelets and statistical pattern recognition. Biomedical Engineering: Applications, Basis and Communications 2014;26.

[126] Coifman RR, Wickerhauser MV. Entropy-based algorithms for best basis selection. Information Theory, IEEE Transactions on 1992;38:713-8.

[127] Kannathal N, Choo ML, Acharya UR, Sadasivan P. Entropies for detection of epilepsy in EEG. Computer methods and programs in biomedicine 2005;80:187-94.

[128] Daniel WW. Biostatistics: a foundation for analysis in the health sciences. New York, USA 1995.

[129] Turan T, Esel E, Karaaslan F, Basturk M, Oguz A, Yabanoglu I. Auditory event-related potentials in panic and generalised anxiety disorders. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2002;26:123-6.

[130] Wang J, Miyazato H, Randall M, Hokama H, Hiramatsu K-I, Ogura C. The N200 abnormalities of auditory event-related potentials in patients with panic disorder. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2003;27:1013-21.

[131] Peng H, Hu B, Liu Q, Dong Q, Zhao Q, Moore P. User-centered depression prevention: An EEG approach to pervasive healthcare. Pervasive Computing Technologies for Healthcare (PervasiveHealth), 2011 5th International Conference on: IEEE; 2011. p. 325-30.

[132] Sammler D, Grigutsch M, Fritz T, Koelsch S. Music and emotion: electrophysiological correlates of the processing of pleasant and unpleasant music. Psychophysiology 2007;44:293-304

APPENDICES

APPENDIX A

Bilgilendirilmiş Onam Formu

Bezmialem Vakıf Üniversitesi Tıp Fakültesi Hastanesi'nde "Panik bozukluk tanısına yönelik elektrofizyolojik parametrelerin mühendislik yöntemleriyle değerlendirilmesi ve psikiyatrik ölçeklerle ilişkilendirilmesi" isimli tez çalışması kapsamında 60 katılımcıdan bazı elektrofizyolojik kayıtlar alınacaktır.Çalışma kapsamında EEG ölçümleri alınacaktır. İşitsel uyaran olarak müzik ve siren sesi kullanılacaktır.Bu çalışma esnasında hiçbir girişimsel işlemde bulunulmayacak ve herhangi bir ilaç verilmeyecektir. Ayrıca kişisel bilgi formu ve iki adet psikiyatrik değerlendirme ölçeği de uygulanacak.

Çalışma kapsamında elde edilen tüm verilerin ve katılımcıların isimlerinin gizli tutulacağı, bilimsel bir amaçla bu verilerin toplandığı ve sadece bilimsel çalışma kapsamında kullanılacaktır.)

Bilgilendirilmiş Gönüllü Olur Formundaki tüm açıklamaları okudum. Bana, yukarıda konusu ve amacı belirtilen araştırma ile ilgili yazılı ve sözlü açıklama Pınar KARAMIKOĞLU tarafından yapıldı ve yapılacak olan araştırma sonrasında herhangi bir sorun ya da sorular olduğu zaman ,araştırmayı yapan Pınar KARAMIKOĞLU'na telefon veya e-mail adresinden ulaşabileceğim bana bildirildi.

(Tel no: 05079514525 e-mail: pnrkaramikoglu@hotmail.com)Araştırmaya gönüllü olarak katıldığımı, istediğim zaman gerekçeli veya gerekçesiz olarak araştırmadan hiç bir hakkımı kaybetmeksizin ayrılabileceğimi biliyorum.Söz konusu araştırmaya,hiçbir baskı ve zorlama olmaksızın kendi rızam ile katılmayı kabul ediyorum.

Katılımcı:	Araştırmacı:
Tarih:	Tarih:
İmza:	İmza:

APPENDIX B

SOSYODEMOGRAFİK ÖZELLİKLER ANKET FORMU

HASTA ADI/SOYADI:

CİNSİYETİ : **DOĞUM YERİ/YILI :**

EĞİTİM DURUMU: mezuniyet yılı: **mezun olduğu okul?**

HAMİLE OLMA VEYA EMZİRME DÖNEMİ DURUMU:

SON MENSTRÜASYON TARİHİNİZ? (bayanlar için)

ALKOL KULLANIM DURUMU:(bir hafta içerisinde kullananlar alınmayacak)

SİGARA KULLANIM DURUMU:paket/gün?

KAÇ YILDIR BU RAHATSIZLIĞINIZ VAR? / HERHANGİ BİR TEDAVİ UYGULANDI MI?

BAŞKA BİR PSİKİYATRİK RAHATSIZLIĞINIZ VAR MI?

HİÇ EKT YAPILDI MI? (YAPILDIYSA NE KADAR SÜRE ÖNCEYDİ?)

(6 ay içerisinde yaptıranlar alınmayacak)

SON 1 HAFTA İÇERİSİNDE ETKEN MADDESİ BENZODİAZEPİN VE SON 3 AYDIR ANTİPSİKOTİK OLAN İLAÇLAR KULLANDINIZ MI?

(diazem, valium, xanax, nervium, ativan, rivotril vb. kullanıyorsa alınmayacak.)

AİLENİZDE NÖROLOJİK YA DA PSİKOLOJİK BİR RAHATSIZLIĞI OLAN VAR MI?

BAŞKA CİDDİ BİR RAHATSIZLIĞINIZ VAR MI? (KALP RAHATSIZLIĞI, DİYABET, TANSİYON)

KULLANDIĞINIZ DİĞER İLAÇLAR NELERDİR?

BAŞKA NÖROLOJİK HASTALIK DURUMU:

APPENDIX C

Beck Anksiyete Ölçeği

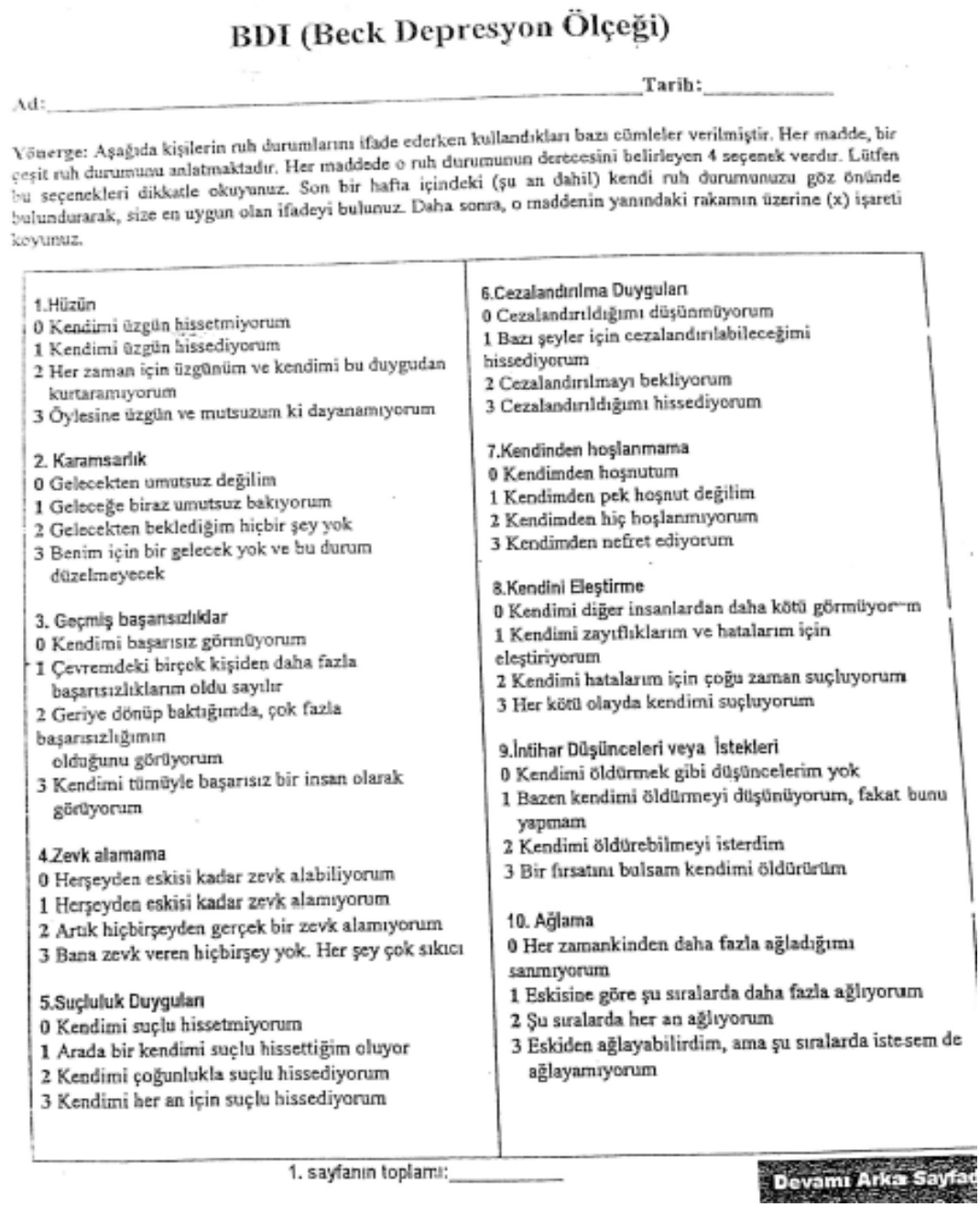
Hastanın Soyadı, Adı:..... Tarih:.....

Aşağıda insanların kaygılı ya da endişeli oldukları zamanlarda yaşadıkları bazı belirtiler verilmiştir.
Lütfen her maddeyi dikkatle okuyunuz. Daha sonra, her maddedeki belirtinin **BUGÜN DAHİL SON BİR (1) HAFTADIR** sizi ne kadar rahatsız ettiğini yandakine uygun yere (x) işareti koyarak belirleyiniz.

	Hiç	Hafif düzeyde Beni pek etkilemedi	Orta düzeyde Hoş değildi ama katlanabildim	Ciddi düzeyde Dayanmakta çok zorlandım
1. Bedeninizin herhangi bir yerinde uyuşma veya karıncalanma				
2. Sıcak/ ateş basmaları				
3. Bacaklarda halsizlik, titreme				
4. Gevşeyememe				
5. Çok kötü şeyler olacak korkusu				
6. Baş dönmesi veya sersemlik				
7. Kalp çarpıntısı				
8. Dengeyi kaybetme duygusu				
9. Dehşete kapılma				
10. Sinirlilik				
11. Boğuluyormuş gibi olma duygusu				
12. Ellerde titreme				
13. Titreklik				
14. Kontrolü kaybetme korkusu				
15. Nefes almada güçlük				
16. Ölüm korkusu				
17. Korkuya kapılma				
18. Mildede hazımsızlık ya da rahatsızlık hissi				
19. Baygınlık				
20. Yüzün kızarması				
21. Terleme (sıcaklığa bağlı olmayan)				

Toplam BECK-A skoru:.....

APPENDIX D



BDI (Beck Depresyon Ölçeği)

Ad:_____ Tarih:_____

Yönerge: Aşağıda kişilerin ruh durumlarını ifade ederken kullandıkları bazı cümleler verilmiştir. Her madde, bir çeşit ruh durumunu anlatmaktadır. Her maddede o ruh durumunun derecesini belirleyen 4 seçenek verdir. Lütfen bu seçenekleri dikkatle okuyunuz. Son bir hafta içindeki (şu an dahil) kendi ruh durumunuzu göz önünde bulundurarak, size en uygun olan ifadeyi bulunuz. Daha sonra, o maddenin yanındaki rakamın üzerine (x) işareti koyunuz.

1.Hüzün
0 Kendimi üzgün hissetmiyorum
1 Kendimi üzgün hissediyorum
2 Her zaman için üzgünüm ve kendimi bu duygudan kurtaramıyorum
3 Öylesine üzgün ve mutsuzum ki dayanamıyorum

2. Karamsarlık
0 Gelecekten umutsuz değilim
1 Geleceğe biraz umutsuz bakıyorum
2 Gelecekten beklediğim hiçbir şey yok
3 Benim için bir gelecek yok ve bu durum düzelmeyecek

3. Geçmiş başarısızlıklar
0 Kendimi başarısız görmüyorum
1 Çevremdeki birçok kişiden daha fazla başarısızlıklarım oldu sayılır
2 Geriye dönüp baktığımda, çok fazla başarısızlığımın olduğunu görüyorum
3 Kendimi tümüyle başarısız bir insan olarak görüyorum

4.Zevk alamama
0 Herşeyden eskisi kadar zevk alabiliyorum
1 Herşeyden eskisi kadar zevk alamıyorum
2 Artık hiçbirşeyden gerçek bir zevk alamıyorum
3 Bana zevk veren hiçbirşey yok. Her şey çok sıkıcı

5.Suçluluk Duyguları
0 Kendimi suçlu hissetmiyorum
1 Arada bir kendimi suçlu hissettiğim oluyor
2 Kendimi çoğunlukla suçlu hissediyorum
3 Kendimi her an için suçlu hissediyorum

6.Cezalandırılma Duyguları
0 Cezalandırıldığımı düşünmüyorum
1 Bazı şeyler için cezalandırılabileceğimi hissediyorum
2 Cezalandırılmayı bekliyorum
3 Cezalandırıldığımı hissediyorum

7.Kendinden hoşlanmama
0 Kendimden hoşnutum
1 Kendimden pek hoşnut değilim
2 Kendimden hiç hoşlanmıyorum
3 Kendimden nefret ediyorum

8.Kendini Eleştirme
0 Kendimi diğer insanlardan daha kötü görmüyorum
1 Kendimi zayıflıklarım ve hatalarım için eleştiriyorum
2 Kendimi hatalarım için çoğu zaman suçluyorum
3 Her kötü olayda kendimi suçluyorum

9.İntihar Düşünceleri veya İstekleri
0 Kendimi öldürmek gibi düşüncelerim yok
1 Bazen kendimi öldürmeyi düşünüyorum, fakat bunu yapmam
2 Kendimi öldürebilmeyi isterdim
3 Bir fırsatını bulsam kendimi öldürürüm

10. Ağlama
0 Her zamankinden daha fazla ağladığımı sanmıyorum
1 Eskisine göre şu sıralarda daha fazla ağlıyorum
2 Şu sıralarda her an ağlıyorum
3 Eskiden ağlayabilirdim, ama şu sıralarda istesem de ağlayamıyorum

1. sayfanın toplamı:_____

Devamı Arka Sayfa

11.Sinirlilik
0 Her zamankinden daha sinirli değilim
1 Her zamankinden daha kolayca sinirleniyor ve kızıyorum
2 Çoğu zaman sinirliyim
3 Eskiden sinirlendiğim şeylere bile artık sinirlenemiyorum

12.İlgi kaybı
0 Diğer insanlara karşı ilgimi kaybetmedim
1 Eskisine göre insanlarla daha az ilgiliyim
2 Diğer insanlara karşı ilgimin çoğunu kaybettim
3 Diğer insanlara karşı hiç ilgim kalmadı

13.Kararsızlık
0 Kararlarımı eskisi kadar kolay ve rahat verebiliyorum
1 Şu sıralar kararlarımı vermeyi erteliyorum
2 Kararlarımı vermekte oldukça güçlük çekiyorum
3 Artık hiç karar veremiyorum

14.Dış Görünüm
0 Dış görünüşümün eskisinden daha kötü olduğunu sanmıyorum
1 Yaşlandığımı ve çekiciliğimi kaybettiğimi düşünüyor ve üzülüyorum
2 Dış görünüşümde artık değiştirilmesi mümkün olmayan olumsuz değişiklikler olduğunu hissediyorum
3 Çok çirkin olduğumu düşünüyorum

15.Çalışma
0 Eskisi kadar iyi çalışabiliyorum
1 Bir işe başlayabilmek için eskisine göre kendimi daha fazla zorlamam gerekiyor
2 Hangi iş olursa olsun, yapabilmek için kendimi çok zorluyorum
3 Hiçbir iş yapamıyorum

16.Uyku düzeninde değişiklik
0 Eskisi kadar rahat uyuyabiliyorum
1 Şu sıralarda eskisi kadar rahat uyuyamıyorum
2 Eskisine göre 1 veya 2 saat erken uyanıyor ve tekrar uyumakta zorluk çekiyorum
3 Eskisine göre çok erken uyanıyor ve tekrar uyumakta zorluk çekiyorum

17.Kolay yorulma
0 Eskisine kıyasla daha çabuk yorulduğumu sanmıyorum
1 Eskisinden daha çabuk yoruluyorum
2 Şu sıralarda neredeyse her şey beni yoruyor
3 Öyle yorgunum ki hiç bir şey yapamıyorum

18.İştahta Değişiklik
0 İştahım eskisinden pek farklı değil
1 İştahım eskisi kadar iyi değil
2 Şu sıralarda iştahım epey kötü
3 Artık hiç iştahım yok

19.Kilo Kaybı
0 Son zamanlarda pek fazla kilo kaybettiğimi sanmıyorum
1 Son zamanlarda istemediğim halde üç kilodan fazla kaybettim
2 Son zamanlarda istemediğim halde beş kilodan fazla kaybettim
3 Son zamanlarda istemediğim halde yedi kilodan fazla kaybettim
Daha az yemeğe çalışarak kilo kaybetmeye çalışıyoru
Evet ( ) Hayır ( )

20.Sağlık Endişesi
0 Sağlığım beni pek endişelendirmiyor
1 Son zamanlarda ağrı, sızı, mide bozukluğu, kabızlık gibi sorunlarım var
2 Ağrı, sızı gibi bu sıkıntılarım beni epey endişelendirdiği için başka şeyleri düşünmek zor geliyor
3 Bu tür sıkıntılar beni öylesine endişelendiriyor ki, ar başka hiçbir şey düşünemiyorum

21.Cinsel İsteğin Kaybolması
0 Son zamanlarda cinsel yaşantımda dikkatimi çeken ş şey yok
1 Eskisine oranla cinsel konularla daha az ilgileniyor
2 Şu sıralarda cinsellikle pek ilgili değilim
3 Artık cinsellikle hiç bir ilgim kalmadı

Sayfa 1'in toplamı:___ Sayfa 2' nin toplamı: ___ = Toplam skor _____

APPENDIX E

BEZMİALEM VAKIF ÜNİVERSİTESİ KLİNİK ARAŞTIRMALARI ETİK KURULU KARAR FORMU

SAYI : 71306642/050-01-04/249 11.12.2013

KONU: Etik Kurulu Kararı

ETİK KURULA İLİŞKİN BİLGİLER		
	ETİK KURULUN ADI	Bezmialem Vakıf Üniversitesi Klinik Araştırmalar Etik Kurulu
	AÇIK ADRESİ	Adnan Menderes Bulvarı Vatan caddesi 34093 Fatih/İstanbul
	TELEFON	(0212) 523 22 88 - 1028
	FAKS	(0212) 533 23 26
	E-POSTA	etikkurulu@bezmialem.edu.tr

BAŞVURU BİLGİLERİ					
	ARAŞTIRMANIN AÇIK ADI	Panik Bozukluğu Tanısına Yönelik Elektrofizyolojik Parametrelerin (Elektroensefalografik Sinyaller) İleri Mühendislik Yöntemleriyle Değerlendirilmesi ve Psikiyatrik Ölçeklerle İlişkilendirilmesi			
	ARAŞTIRMA PROTOKOL KODU				
	KOORDİNATÖR/SORUMLU ARAŞTIRMACI UNVANI/ADI/SOYADI	Prof. Dr. İsmet KIRPINAR			
	KOORDİNATÖR/SORUMLU ARAŞTIRMACININ UZMANLIK ALANI	Ruh Sağlığı ve Hastalıkları			
	KOORDİNATÖR/SORUMLU ARAŞTIRMACININ BULUNDUĞU MERKEZ	Bezmialem Vakıf Üniversitesi Tıp Fakültesi Hastanesi			
	DESTEKLEYİCİ	Fatih Üniversitesi BAP Birimi			
	DESTEKLEYİCİNİN YASAL TEMSİLCİSİ				
	ARAŞTIRMANIN FAZI VE TÜRÜ	FAZ 1	☐		
		FAZ 2	☐		
		FAZ 3	☐		
		FAZ 4	☐		
		Gözlemsel ilaç çalışması	☐		
		İlaç dışı klinik araştırma (akademik amaçlı)	☒ Tanı kriterleri oluşturmak		
		Diğer ise belirtiniz:			
	ARAŞTIRMAYA KATILAN MERKEZLER	TEK MERKEZ ☒	ÇOK MERKEZLİ ☐	ULUSAL ☐	ULUSLARARASI ☐

	Belge Adı	Tarihi	Versiyon Numarası	Dili
DEĞERLENDİRİLEN BELGELER	ARAŞTIRMA PROTOKOLÜ	15.12.2013	-	Türkçe ☒ İngilizce ☐ Diğer ☐
	BİLGİLENDİRİLMİŞ GÖNÜLLÜ OLUR FORMU	-	-	Türkçe ☒ İngilizce ☐ Diğer ☐
	OLGU RAPOR FORMU			Türkçe ☐ İngilizce ☐ Diğer ☐
	ARAŞTIRMA BROŞÜRÜ			Türkçe ☐ İngilizce ☐ Diğer ☐
DEĞERLENDİRİLEN DİĞER BELGELER	Belge Adı		Açıklama	
	SİGORTA	☐		
	ARAŞTIRMA BÜTÇESİ	☒		
	BİYOLOJİK MATERYEL TRANSFER FORMU	☐		

BEZMİALEM VAKIF ÜNİVERSİTESİ KLİNİK ARAŞTIRMALARI ETİK KURULU KARAR FORMU

	İLAN	☐	
	YILLIK BİLDİRİM	☐	
	SONUÇ RAPORU	☐	
	GÜVENLİLİK BİLDİRİMLERİ	☐	
	DİĞER:	☒	- Sorumlu araştırmacı ve yardımcı araştırmacılara ait özgeçmiş formları - Çalışmanın Helsinki Bildirgesi, İKUİİLU'ya uygun yürütüleceğine dair taahhütname - Araştırma ile ilgili yayınlar
KARAR BİLGİLERİ	**Karar No: 47 / 14**	**Tarih: 11.12.2013**	
	Yukarıda bilgileri verilen başvuru dosyası ile ilgili belgeler araştırmanın/çalışmanın gerekçe, amaç, yaklaşım ve yöntemleri dikkate alınarak incelenmiş ve uygun bulunmuş olup araştırmanın/çalışmanın başvuru dosyasında belirtilen merkezlerde gerçekleştirilmesinde etik ve bilimsel sakınca bulunmadığına toplantıya katılan etik kurul üye tam sayısının salt çoğunluğu ile karar verilmiştir. Klinik Araştırmalar Hakkında Yönetmelik kapsamında yer alan araştırmalar/çalışmalar için Türkiye İlaç ve Tıbbi Cihaz Kurumu'ndan izin alınması gerekmektedir.		

BEZMİALEM VAKIF ÜNİVERSİTESİ KLİNİK ARAŞTIRMALARI ETİK KURULU KARAR FORMU

BEZMİALEM VAKIF ÜNİVERSİTESİ KLİNİK ARAŞTIRMALARI ETİK KURULU	
ETİK KURULUN ÇALIŞMA ESASI	Klinik Araştırmalar Hakkında Yönetmelik, İyi Klinik Uygulamaları Kılavuzu
BAŞKANIN UNVANI / ADI / SOYADI:	Prof. Dr. Reha ERKOÇ

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