

ORIGINAL RESEARCH**Emotion regulation difficulties and cognitive emotional regulation in patients with temporal lobe epilepsy**

Abbas Masjedi Arani¹, Sepideh Batebi¹, Behrooz Dolatshahi*², Mojtaba Azimian³

1. *Department of Clinical Psychology, Faculty of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran.*
2. *Department of Clinical Psychology, University of Social Welfare and Rehabilitation Sciences, Tehran, Iran. ORCID: 0000-0003-2230-9562*
3. *Department of Clinical Sciences, University of Social Welfare and Rehabilitation Sciences, Tehran, Iran.*

*Corresponding Author:

Address: Department of Clinical Psychology, University of Social Welfare and Rehabilitation Sciences, Kodakyar Ave., Daneshjo Blvd., Evin, Tehran, Iran.

Email: dolatshahee@yahoo.com

ORCID: 0000-0003-2230-9562

Date Received: June, 2020

Date Accepted: July, 2020

Online Publication: February 28, 2021

Abstract

Objective: Temporal lobe epilepsy (TLE) is one of the most prevalent types of complex partial epilepsy in adults. Due to the damage to the amygdala, patients with TLE struggle with emotional problems. The purpose of this study was to investigate the functions of emotion regulation and cognitive-emotional self-regulation in patients with TLE compared to non-epileptic individuals.

Method: In this study, 80 patients with TLE were recruited based on some inclusion criteria and compared with 80 non-epileptics by considering their emotional functions. Questionnaires for evaluating difficulties in emotion regulation (Gratz & Roemer) and cognitive emotional regulation (Garnefski) were given to the participants of the study. Finally, data analysis was performed using multivariate analysis of variance (MANOVA) by SPSS 19 Statistics.

Results: The results of MANOVA test showed a significant difference between the components of difficulties in emotion regulation as well as cognitive-emotional regulation between patients with TLE and the healthy group ($P < 0.01$).

Conclusion: Patients with TLE had more difficulties in emotion regulation and higher negative cognitive emotional regulation strategies than healthy subjects.

Keywords: Epilepsy, Temporal lobe epilepsy, Emotion, Cognition, Function

Introduction

Epilepsy is one of the most prevalent chronic neurological diseases that include sudden, frequent and abnormal discharges of the brain's nervous tissue [1]. Temporal lobe epilepsy is the most current syndrome of complicated focal seizures can be divided in to mesial and cryptogenic temporal lobe epilepsy [2]. More than a quarter of epileptic patients also experience psychological symptoms that may require distinct treatment. Moreover, more than 10% of patients admitted to the psychiatric wards are epileptic patients [3]. Researches have shown comorbidities such as anxiety and depression in patients with TLE [4-6], so underestimating these mood and anxiety symptoms and late treatment have negative effects on both the patients' quality of life and the efficacy of medications administered for controlling the seizures [7]. One of the psychological variables that is impaired in TLE patients is emotional processing and emotion regulation [8-9]. This impairment is due to the amygdala damage in these patients [10-13]. Actually, the amygdala damage that has effects on emotion control and regulation can affect the social communication of TLE patients [14-16], as well as the exacerbation of related psychological symptoms, such as anxiety and depression [17]. Some studies have expressed the role of the mediating variable of emotion regulation in mental disorders [18-20]. Furthermore, even psychological problems such as anxiety and depression have been attributed to emotional dysregulation [18-23]. Gross [24] and Gratz [25] considered the emotion regulation as the internal and external processes responsible for monitoring, assessing and modifying emotional reactions. Structures in the anterior-medial temporal lobe, especially the amygdala, have crucial role in emotional coding [26]. Patients with mesial temporal lobe epilepsy and the amygdala damage have impaired emotion recognition and processing [8]. Moreover, it has been shown that there are some emotional abnormalities in patients with mesial temporal lobe atrophy [27]. Emotional processing and emotion recognition are also impaired in patients with mesial temporal lobe epilepsy and asymmetrical amygdala damage [28].

Considering what mentioned above, due to the chronicity of TLE and its impact on cognitive and emotional processing in the brain, TLE patients have high behavioral-emotional problems such as depression and anxiety that seriously affect their individual, family and social-occupational performance as well as the quality of life of these patients. To the best of our knowledge, there has been no study on the emotion regulation difficulty and cognitive-emotional regulation in these patients.

One way of protecting TLE patients against emotional problems is to identify emotional dysfunctions, such as emotional and cognitive dysregulation in these patients. This, therefore, in addition to preventing the intensification of emotional symptoms, can be also effective in providing complementary therapies, such as emotion-based psychotherapies. Consequently, the purpose of the present study was to evaluate the difficulties in emotion regulation and cognitive-emotional regulation in TLE patients and compare them with healthy individuals.

Materials and Methods

Eighty patients were recruited during their visits to the Neurology Clinic of Chamran Hospital, Shohadaye Tajrish Hospital and Iranian epilepsy association, after definitive diagnosis of TLE using purposive sampling method. The inclusion criteria were: 1) definitive diagnosis of TLE through clinical examinations by neurologists and electroencephalography; 2) having reading and writing skill; 3) an age between 18 and 60 years. The exclusion criteria were: 1) severe mental and personality disorders; 2) using psychotropic drugs or any other medication with adverse effects affecting the emotions and mood of the patients; 3) drug and alcohol dependence.

A clinical psychologist to rule out severe mental and personality disorders psychologically evaluated all patients. Next, each of 80 patients filled out a written consent form for participating in the study. Finally, the emotional functioning variables (such as emotion regulation difficulty and cognitive-emotional self-regulation) of the patients were compared with that of 80 healthy subjects (without any history of epilepsy and epileptic seizures).

Measurement:

DSM-V-based psychiatric clinical interview: all subjects were psychiatrically interviewed for the diagnosis of severe mental and personality disorders as well as substance-related disorders.

Difficulties in emotion regulation scale (DERS, Gratz & Roemer):

This scale includes 36 items with an overall score and six subscale scores related to the various dimensions of difficulty in emotion regulation. These subscales included non-acceptance of emotions, difficulties engaging in goal-directed behavior, difficulties controlling impulsive behaviors, lack of emotional awareness, limited access to effective strategies and lack of emotional clarity. The items were scored on a 5-point Likert scale. This scale showed adequate internal consistency for the total score (a Cronbach's alpha of 0.93) and for all subscales (alpha greater than 0.80). The test-retest reliability of the scale was also reported suitable over the period [25]. Aminan investigated the reliability of this scale by using Cronbach's alpha and split-half methods, and found the values of 0.86 and 0.80, respectively [29]. In another study the reliability of this scale reported by Alpha value ranged from 0.88 to 0.93 [30].

Cognitive emotion regulation questionnaire (CERQ):

The cognitive emotion regulation questionnaire is a self-report questionnaire that is designed by Garnefski, Karaaij and Spinhoven in 1999, and developed in 2001. This questionnaire is used to identify cognitive coping strategies after experiencing an adverse event. The original version of CERQ is a 36-item questionnaire consisting of nine principal components (Self-blame, Acceptance, Rumination, Positive refocusing, Planning, Putting into perspective, Positive reappraisal, Catastrophizing and Other-blame). In this questionnaire, self-blame, other-blame, rumination and catastrophizing form the negative emotion regulation strategies; furthermore, acceptance, planning, positive refocusing, positive reappraisal and putting into perspective form positive emotion regulation strategies. The developer of this questionnaire reported internal consistency of positive, negative strategies and total score by

Alpha value of 0.91, 0.87, and 0.93 respectively [31]. In Iranian sample, internal consistency by using Cronbach's alpha method obtained 0.82 for all cognitive scale [32].

Results

The results of descriptive study of demographic variables were obtained for TLE patients with an average age of 45 years, a standard deviation of 3.42, predominantly male (75.2%), single (58.1%), high school education or less (61%), unemployed 56.9% and middle or lower socioeconomic statuses (87.5%). In the group of healthy individuals, the average age was 42 years, standard deviation 3.78; moreover they were predominantly male (69.1%), married (53.2%), with academic degrees (63%), employed (52.3%) and of middle or lower socioeconomic statuses (85.7%). There was no significant difference between the two groups in terms of demographic variables.

Descriptive scores of difficulties in emotion regulation in the subjects are listed in Table 1. Based on these results, the mean of all components of difficulties in emotion regulation, including non-acceptance of emotions, difficulties engaging in goal-directed behavior, difficulties controlling impulsive behaviors, lack of emotional awareness, limited access to effective strategies and lack of emotional clarity were higher in TLE patients than non-epileptics.

As well Based on this table, the cognitive-emotion regulation scores for the TLE and non-epileptic subjects, the mean scores of acceptance, planning, positive refocusing, and positive reappraisal and putting into perspective were higher in the healthy individuals than the TLE patients. Moreover, the mean scores of self-blame, other-blame, rumination and catastrophizing as the negative cognitive-emotion regulation strategies were higher in the TLE patients than the healthy individuals.

Table 1. Mean scores and standard deviation of the difficulties in emotion regulation and cognitive emotion regulation subscales in TLE and non-epileptic group

Variables	TLE group		Non-epileptic group	
	Mean	SD	Mean	SD
Non-acceptance emotion	17.26	6.60	11.10	4.12
Goal-directed behavior	15.60	4.85	11.77	4.06
Impulsive behaviors	17.18	6.41	12.00	4.35
Emotional awareness	16.69	4.60	13.43	3.68
Effective strategies	22.65	7.13	14.68	4.69
Emotional clarity	14.18	2.74	10.82	3.09
Self-blame	6.37	4.39	4.77	3.01
Acceptance	7.68	3.87	8.21	3.64
Rumination	8.50	3.46	6.75	2.73
Positive refocusing	9.69	3.50	10.58	3.25
Planning	12.05	3.56	12.13	2.96
Positive reappraisal	11.15	5.62	11.96	3.03
Putting into perspective	8.01	3.39	9.61	3.34
Catastrophizing	7.00	4.84	4.13	3.28
Other-blame	6.50	4.54	4.04	2.95

Kolmogorov-Smirnov test of normality was used for the variables of the study. The non-significance of the test indicated that all studied variables had normal distribution ($P < 0.05$). The results of multivariate analysis of variance (MANOVA) were used to investigate the significance of the difference between the two groups (Table 2). From Table 2, the F ratio was significant at the 99% confidence level. Accordingly, there was a statistically significant difference between the groups at least for one component of difficulties in emotion regulation.

Table 2. The results of MANOVA test of the difficulties in emotion regulation in TLE and non-epileptic group

Effect	Tests	Value	F	Hypothesis df	Error df	P-Value	Partial Eta Squared
Group	Pillai's Trace	0.43	14.26	6	113	0.001	0.431
	Wilk's Lambda	0.56	14.26	6	113	0.001	0.431
	Hotelling's Trace	0.75	14.26	6	113	0.001	0.431
	Roy's Largest Root	0.75	14.26	6	113	0.001	0.431

To investigate the significance of differences in the subscales, we consider the between-subject effects. The results in Table 3 show a significant difference between the two groups for the components of non-acceptance of emotions, difficulties engaging in goal-directed behavior, difficulties controlling impulsive behaviors, lack of emotional awareness, limited access to effective strategies and lack of emotional clarity ($P < 0.01$). In other words, emotion regulation in TLE patients is more dysfunctional than healthy subjects.

Table 3. The results of between-subject effects of the difficulties in emotion regulation in TLE and non-epileptic group

Source	Dependent Variable	Sum of Squares	Df	Mean Square	F	P-Value	Partial Eta Squared
Group	Non-acceptance emotion	1140.83	1	1140.83	37.63	0.001	0.242
	Goal-directed behavior	440.83	1	440.83	21.96	0.001	0.157
	Impulsive behaviors	803.52	1	803.52	26.75	0.001	0.185
	Emotional awareness	318.75	1	318.75	18.33	0.001	0.134
	Effective strategies	1906.58	1	1906.58	52.28	0.001	0.307
	Emotional clarity	339.18	1	339.18	39.72	0.001	0.252
Error	Non-acceptance emotion	3577.13	118	30.31			
	Goal-directed behavior	2368.24	118	20.07			
	Impulsive behaviors	3544.57	118	30.03			
	Emotional awareness	2051.89	118	17.38			
	Effective strategies	4302.54	118	36.46			
	Emotional clarity	1007.61	118	8.53			

The differences between the two groups in terms of cognitive emotion regulation variable were presented using the MANOVA test results (Table 4), which showed the significance of the F-ratio at the 99% confidence level.

Table 4. The results of MANOVA test of cognitive emotion regulation in TLE and non-epileptic group

Effect	Tests	Value	F	Hypothesis df	Error df	P-Value	Partial Eta Squared
Group	Pillai's Trace	0.22	3.52	9	110	0.001	0.224
	Wilk's Lambda	0.77	3.52	9	110	0.001	0.224
	Hotelling's Trace	0.28	3.52	9	110	0.001	0.224
	Roy's Largest Root	0.28	3.52	9	110	0.001	0.224

For examining the significance of differences between the two groups for each of the of cognitive-emotional regulation subscales, the between-subject effects are presented in Table 5. According to these results, the between-subject effects on the cognitive-emotion regulation components including self-blame, rumination, putting into perspective, catastrophizing and other-blame were significant at $P < 0.01$. In other words, these components in patients with TLE were more dysfunctional than in non-epileptic subjects.

Table 5. The results of between subject effects of cognitive emotion regulation in TLE and non-epileptic group

Source	Dependent Variable	Sum of Squares	Df	Mean Square	F	P-Value	Partial Eta Squared	
Group	Self-blame	76.31	1	76.31	5.36	0.022	0.044	
	Acceptance	8.50	1	8.50	0.60	0.440	0.005	
	Rumination	92.09	1	92.09	9.45	0.003	0.074	
	Positive refocusing	14.40	1	14.40	1.26	0.263	0.011	
	Planning	0.17	1	0.17	0.01	0.899	0.000	
	Positive reappraisal	19.53	1	19.53	0.95	0.330	0.008	
	Putting into perspective	76.80	1	76.80	6.76	0.011	0.054	
	Catastrophizing	246.40	1	246.40	14.38	0.000	0.109	
	Other-blame	181.47	1	181.47	12.36	0.001	0.095	
	Error	Self-blame	1677.95	118	14.22			
		Acceptance	1670.09	118	14.15			
		Rumination	1149.46	118	9.74			
		Positive refocusing	1346.24	118	11.40			
		Planning	1266.62	118	10.73			
Positive reappraisal		2407.44	118	20.40				
Putting into perspective		1340.53	118	11.36				
Catastrophizing		2021.58	118	17.13				
Other-blame		1732.52	118	14.68				

Discussion

In the present study, the difficulties in emotion regulation and cognitive-emotional regulation were compared between TLE patients and healthy individuals. For the difficulties in emotion regulation variable, the results showed that the six components of non-acceptance of emotions, difficulties engaging in goal-directed behavior, difficulties controlling impulsive behaviors, lack of emotional awareness, limited access to effective strategies and lack of emotional clarity in the TLE group were more dysfunctional than in the healthy group. Especially, for the three components of limited access to effective strategies, non-acceptance of emotions and lack of emotional clarity, the emotional dysfunctioning was more considerable.

In patients with TLE, electrodermal responses to induced emotions, particularly to fear, were lower than non-epileptics [17]. Actually, the amygdala is responsible for controlling emotional processing in this case [8-9]. In TLE, damage to the amygdala, limbic system [10-13] and hypothalamic pituitary adrenal (HPA) axis dysfunction have been proven [33]. Therefore, due to the role of the amygdala and insula as the key structures in

emotion regulation [34-36], and the connection between these two structures and the other areas of the brain, such as medial and ventrolateral regions of the prefrontal cortex and anterior cingulate cortex [34], as well problems in bilateral amygdala activity during emotion regulation [37] the difficulties in emotion regulation can be observed in TLE patients. The results of the present study on difficulties in emotion regulation showed that all emotional dysregulation components, especially limited access to effective strategies, non-acceptance of and lack of emotional clarity were common in these patients.

Functional impairment in cognitive-emotional regulation in TLE patients was more than that in non-epileptics. Findings showed that, for the components of self-blame, rumination, putting into perspective, catastrophizing and other-blame, the patients with TLE had more functional impairment than the healthy group. Actually, the difference with the healthy group was in the three components of catastrophizing, other-blame and rumination was greater than the other two components of self-blame and putting into perspective.

Previous studies have found that temporal lobe damage leads to changes in social-emotional behavior. These studies have expressed that the amygdala acts as a neuronal substrate in social cognition and that damage to the amygdala leads to behavioral changes [38]. Additionally, it has been found that the mesolimbic circuitry is associated with social cognition and perception [39-40], and the social perception is impaired in patients with mesial temporal lobe epilepsy [27, 41-43].

The role of the amygdala in cognitive-emotional regulation functions including perception, attention, learning, memory and decision making has been shown. Moreover, the involvement of the amygdala in investigating emotional events and providing appropriate responses to these events has been well studied [44].

In the study of Yamada [45], the role of abnormal circuitry in hyper excitability of epilepsy with amygdala hypersensitivity and the role of limbic system excitability in

cognitive-emotional processing have been demonstrated. The role of the amygdala as a principal structure of emotional processing in the cognitive reappraisal of negative emotions (one of the cognitive-emotional regulation techniques) has been investigated [37].

Aas [46] stated that the amygdala is important for excellent cognitive function in humans and a potential neural mediator between stress and cognitive change in humans. It was also shown that rumination (as a maladaptive emotion regulation strategy) is related to an increased association between the bilateral region of the amygdala and the anterior cingulate cortex.

As mentioned above, due to the damage to the amygdala, its dysfunction and amygdala hypometabolism in TLE patients [47-50], attributional biases towards negative stimuli, which leads to difficulties in disengaging their attention from negative stimuli [51-53].

The temporal lobe and the amygdala have an important role in the expression and regulation of emotions, particularly in stress and threatening stimuli. Indeed, it has been accepted that epileptic seizures are not accidental, and they occur in response to environmental stimuli. Patients with TLE pay close attention to negative stress stimuli and anxiety that also lead to epileptic seizures [47,51]. Therefore, according to the literature as mentioned above and the findings of the present study, TLE patients mostly use maladaptive cognitive-emotional regulation strategies.

The results of this study showed that TLE patients have more difficulties in emotion regulation as well as more negative cognitive-emotional regulation strategies than non-epileptics.

Acknowledgment

We would like to especially thank all the staff who helped us to conduct this research, and in particular, the neuroscientists of Chamran Hospital, Shohadaye Tajrish Hospital and Iranian Epilepsy Association.

Conflict of interest

Authors declare no conflict of interest.

References:

1. Borbely K. Functional imaging (PET and SPECT) in epilepsy. *Orvosi hetilap*. 2001; 142(44):2405-14.
2. Engel Jr J. Introduction to temporal lobe epilepsy. *Epilepsy research*. 1996; 26(1):141-50.
3. Javaheri R, Neshat-Doost HT, Molavi H, Zare M. Efficacy of cognitive-behavioral stress management therapy on improving the quality of life in females with temporal lobe epilepsy. *Arak Medical University Journal*. 2010; 13(2): 32-43.
4. Altshuler LL, Devinsky O, Post RM, Theodore W. Depression, anxiety, and temporal lobe epilepsy: laterality of focus and symptoms. *Archives of Neurology*. 1990; 47(3):284-8.
5. Reuber M, ANDERSEN B, Elger CE, Helmstaedter C. Depression and anxiety before and after temporal lobe epilepsy surgery. *Seizure*. 2004; 13(2):129-35.
6. Swinkels WA, van Emde Boas W, Kuyk J, Van Dyck R, Spinhoven P. Interictal depression, anxiety, personality traits, and psychological dissociation in patients with temporal lobe epilepsy (TLE) and extra-TLE. *Epilepsia*. 2006; 47(12):2092-103.
7. Cramer JA, Brandenburg N, Xu X. Differentiating anxiety and depression symptoms in patients with partial epilepsy. *Epilepsy & Behavior*. 2005; 6(4):563-9.
8. Wendling AS, Hirsch E, Wisniewski I, Davanture C, Ofer I, Zentner J, Bilic S, Scholly J, Staack AM, Valenti MP, Schulze-Bonhage A. Selective amygdalohippocampectomy versus standard temporal lobectomy in patients with mesial temporal lobe epilepsy and unilateral hippocampal sclerosis. *Epilepsy research*. 2013; 104(1-2):94-104.
9. Vaugier L, Aubert S, McGonigal A, Trebuchon A, Guye M, Gavaret M, Regis J, Chauvel P, Wendling F, Bartolomei F. Neural networks underlying hyperkinetic seizures of "temporal lobe" origin. *Epilepsy research*. 2009; 86(2-3):200-8.
10. Goldin PR, McRae K, Ramel W, Gross JJ. The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biological psychiatry*. 2008; 63(6):577-86.
11. Fowler HL, Baker GA, Tipples J, Hare DJ, Keller S, Chadwick DW, Young AW. Recognition of emotion with temporal lobe epilepsy and asymmetrical amygdala damage. *Epilepsy & Behavior*. 2006; 9(1):164-72.
12. Bernasconi N, Bernasconi A, Caramanos Z, Antel SB, Andermann F, Arnold DL. Mesial temporal damage in temporal lobe epilepsy: a volumetric MRI study of the hippocampus, amygdala and parahippocampal region. *Brain*. 2003; 126(2):462-9.
13. Pitkänen A, Tuunanen J, Kälviäinen R, Partanen K, Salmenperä T. Amygdala damage in experimental and human temporal lobe epilepsy. *Epilepsy research*. 1998; 32(1-2):233-53.
14. Brothers L. The neural basis of primate social communication. *Motivation and emotion*. 1990; 14(2):81-91.
15. Bonora A, Benuzzi F, Monti G, Mirandola L, Pugnaghi M, Nichelli P, Meletti S. Recognition of emotions from faces and voices in medial temporal lobe epilepsy. *Epilepsy & Behavior*. 2011; 20(4):648-54.
16. Haxby JV, Hoffman EA, Gobbini MI. Human neural systems for face recognition and social communication. *Biological psychiatry*. 2002; 51(1):59-67.
17. Kotwas I, McGonigal A, Khalfa S, Bastien-Toniazzo M, Bartolomei F, Micoulaud-Franchi JA. A case-control study of skin conductance biofeedback on seizure frequency and emotion regulation in drug-resistant temporal lobe epilepsy. *International Journal of Psychophysiology*. 2018; 123:103-10.
18. Gross JJ, Muñoz RF. Emotion regulation and mental health. *Clinical psychology: Science and practice*. 1995; 2(2):151-64.

19. Sheppes G, Suri G, Gross JJ. Emotion regulation and psychopathology. *Annual review of clinical psychology*. 2015; 11:379-405.
20. Werner K, Gross J J. Emotion regulation and psychopathology: A conceptual framework. In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment*. The Guilford Press 2010;13–37.
21. Mennin DS, McLaughlin KA, Flanagan TJ. Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. *Journal of anxiety disorders*. 2009; 23(7):866-71.
22. Amstadter A. Emotion regulation and anxiety disorders. *Journal of anxiety disorders*. 2008; 22(2):211-21.
23. Salters-Pedneault K, Roemer L, Tull MT, Rucker L, Mennin DS. Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy and Research*. 2006; 30(4):469-80.
24. Gross JJ, Thompson RA. Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation*. The Guilford Press 2007; 3–24.
25. Gratz KL, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of psychopathology and behavioral assessment*. 2004; 26(1):41-54.
26. Gomez-Ibañez A, Urrestarazu E, Viteri C. Recognition of facial emotions and identity in patients with mesial temporal lobe and idiopathic generalized epilepsy: an eye-tracking study. *Seizure*. 2014; 23(10):892-8.
27. Reynders HJ, Broks P, Dickson JM, Lee CE, Turpin G. Investigation of social and emotion information processing in temporal lobe epilepsy with ictal fear. *Epilepsy & Behavior*. 2005; 7(3):419-29.
28. Bonora A, Benuzzi F, Monti G, Mirandola L, Pugnaghi M, Nichelli P, Meletti S. Recognition of emotions from faces and voices in medial temporal lobe epilepsy. *Epilepsy & Behavior*. 2011; 20(4):648-54.
29. Soltan Mohammadlou S, Gharraee B, Fathali Lavasani F, Gohari MR. The relationship of Behavioral Activation and Inhibition Systems (BAS/BIS), difficulty of emotional regulation, metacognition with worry. *Research in cognitive and behavioral sciences* 2014; 2(5): 85-100.
30. Dehghani Y, Moradi N, Tabnak F, Afshin SA. Investigating the relationship between humor and difficulty in regulation of emotions and alexithymia in students. *Journal of Fundamentals of Mental Health*. 2018; 20(2):113-20.
31. Garnefski N, Kraaij V. The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment*. 2007; 23(3):141-9.
32. Essazadeghan A, Hassani M, Ahmadian L, Amani J. Comparison of Cognitive Emotion Regulation Strategies and General Health Characteristics of Individuals with and without Alexithymia. *Psychological research* 2013;16(1):65-83.
33. Maguire J, Salpekar JA. Stress, seizures, and hypothalamic–pituitary–adrenal axis targets for the treatment of epilepsy. *Epilepsy & Behavior*. 2013; 26(3):352-62.
34. Phillips ML, Drevets WC, Rauch SL, Lane R. Neurobiology of emotion perception I: The neural basis of normal emotion perception. *Biological psychiatry*. 2003; 54(5):504-14.
35. Price JL. Comparative aspects of amygdala connectivity. *Annals of the New York Academy of Sciences*. 2003; 985(1):50-8.
36. Bookheimer S. Functional MRI of language: new approaches to understanding the cortical organization of semantic processing. *Annual review of neuroscience*. 2002; 25(1):151-88.

37. Sarkheil P, Zilverstand A, Kilian-Hütten N, Schneider F, Goebel R, Mathiak K. fMRI feedback enhances emotion regulation as evidenced by a reduced amygdala response. *Behavioural Brain Research*. 2015; 281:326-32.
38. Amaral DG, Bauman MD, Capitanio JP, Lavenex P, Mason WA, Mauldin-Jourdain ML, Mendoza SP. The amygdala: is it an essential component of the neural network for social cognition? *Neuropsychologia*. 2003; 41(4):517-22.
39. Fournier NM, Calverley KL, Wagner JP, Poock JL, Crossley M. Impaired social cognition 30 years after hemispherectomy for intractable epilepsy: the importance of the right hemisphere in complex social functioning. *Epilepsy & Behavior*. 2008; 12(3):460-71.
40. Marin JC, Moura PJ, Cysneiros RM, Colugnati DB, Cavalheiro EA, Scorza FA, Xavier GF, Zilbovicius M, Mercadante MT. Temporal lobe epilepsy and social behavior: an animal model for autism?. *Epilepsy & Behavior*. 2008; 13(1):43-6.
41. Schacher M, Winkler R, Grunwald T, Kraemer G, Kurthen M, Reed V, Jokeit H. Mesial temporal lobe epilepsy impairs advanced social cognition. *Epilepsia*. 2006; 47(12):2141-6.
42. Broicher SD, Kuchukhidze G, Grunwald T, Krämer G, Kurthen M, Jokeit H. "Tell me how do I feel?"—Emotion recognition and theory of mind in symptomatic mesial temporal lobe epilepsy. *Neuropsychologia*. 2012; 50(1):118-28.
43. Tanaka A, Akamatsu N, Yamano M, Nakagawa M, Kawamura M, Tsuji S. A more realistic approach, using dynamic stimuli, to test facial emotion recognition impairment in temporal lobe epilepsy. *Epilepsy & Behavior*. 2013; 28(1):12-6.
44. Patin A, Hurlemann R. Modulating amygdala responses to emotion: Evidence from pharmacological fMRI. *Neuropsychologia*. 2011; 49(4):706-17.
45. Yamada M, Murai T, Sato W, Namiki C, Miyamoto T, Ohigashi Y. Emotion recognition from facial expressions in a temporal lobe epileptic patient with ictal fear. *Neuropsychologia*. 2005; 43(3):434-41.
46. Aas M, Navari S, Gibbs A, Mondelli V, Fisher HL, Morgan C, Morgan K, MacCabe J, Reichenberg A, Zanelli J, Fearon P. Is there a link between childhood trauma, cognition, and amygdala and hippocampus volume in first-episode psychosis? *Schizophrenia research*. 2012; 137(1-3):73-9.
47. Lanteaume L, Guedj E, Bastien-Toniazzo M, Magalahaes A, Mundler O, Bartolomei F. Cognitive and metabolic correlates of emotional vulnerability in patients with temporal lobe epilepsy. *J Neurol Neurosurg Psychiatry*. 2012; 83(5):522-8.
48. Richardson EJ, Griffith HR, Martin RC, Paige AL, Stewart CC, Jones J, Hermann BP, Seidenberg M. Structural and functional neuroimaging correlates of depression in temporal lobe epilepsy. *Epilepsy & Behavior*. 2007; 10(2):242-9.
49. Kondziella D, Alvestad S, Vaaler A, Sonnewald U. Which clinical and experimental data link temporal lobe epilepsy with depression? *Journal of neurochemistry*. 2007; 103(6):2136-52.
50. Takaya S, Ikeda A, Mitsueda-Ono T, Matsumoto R, Inouchi M, Namiki C, Oishi N, Mikuni N, Ishizu K, Takahashi R, Fukuyama H. Temporal lobe epilepsy with amygdala enlargement: a morphologic and functional study. *Journal of Neuroimaging*. 2014; 24(1):54-62.
51. Lanteaume L, Bartolomei F, Bastien-Toniazzo M. How do cognition, emotion, and epileptogenesis meet? A study of emotional cognitive bias in temporal lobe epilepsy. *Epilepsy & Behavior*. 2009; 15(2):218-24.
52. Vuilleumier P. How brains beware: neural mechanisms of emotional attention. *Trends in cognitive sciences*. 2005; 9(12):585-94.
53. Vuilleumier P, Richardson MP, Armony JL, Driver J, Dolan RJ.

Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature neuroscience*. 2004; 7(11):1271.