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Cognitive and perceptual functions in patients with occipital lobe epilepsy, patients with migraine, and healthy controls

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ABSTRACT

This study was conducted to compare cognitive and perceptual functions among patients with occipital lobe epilepsy, patients with migraine, and healthy individuals, in relation to the moderating roles of gender and educational level. Participants included 93 individuals from Mashhad City, Khorasan-e-Razavi province, Iran. A demographic questionnaire and Bender–Gestalt II (BGT-II; Brannigan & Decker, 2003) were used for data collection in this study. Results showed significant group differences for copy, recall, motor, and perceptual subscales of BGT-II in these samples, where patients with occipital lobe epilepsy and patients with migraine having significantly lower scores than healthy individuals. Also, patients with occipital lobe epilepsy had significantly poorer scores in all subscales of the BGT-II in comparison with the patients with migraine. There were no significant differences with regard to gender and educational level when considering dependent variables in the present study.

1. Introduction

Different relationships exist between epileptic seizures and headaches, which have given rise to unclear or controversial terminologies in this field of study [1]. Epilepsy and migraine are both common episodic disorders; and both diseases have many shared clinical features and causal pathophysiological mechanisms [2]. Both epilepsy and migraine are considered as paroxysmal and chronic diseases where pathophysiological and clinical symptomatology overlaps, particularly with regard to visual and other sensory disturbances, pain, and alterations of consciousness. Epilepsy and migraine have a similar genetic predisposition, as well as pathophysiological mechanisms. There is a substantial connection between epilepsy and migraine across various aspects, including classification, clinical features, epidemiology, genetics, pathophysiology, and treatment [3]. However, alterations of consciousness in migraine are rare and limited to basilar migraine [4].

Although there is no conclusive evidence of a real causal relationship between epilepsy and migraine, the association of these episodic neurological disorders with neurocognitive functions is an interesting subject, which has not been substantially investigated in the field of psychological research. Evidence shows that psychological impacts are greater in epilepsy and extend beyond the neurological condition itself, while in the case of migraines, the psychological impact does not appear to extend further than the neurological condition [5]. Therefore, this study is aimed to investigate the cognitive and perceptual functions with regards the moderating roles of gender and the levels of education in patients with occipital lobe epilepsy (OLE), patients with migraine, and healthy controls.

Epilepsy can significantly interrupt normal cognitive and perceptual functions in adults [6,7]. Cognitive deficits in epilepsy may be cured using antiepileptic drugs or surgery, and by treatment of comorbid disorders such as depression [8], but some of the negative side effects of antiepileptic medications are their impact on the speed of information processing and attention [9]. Among the comorbidities associated with epilepsy, cognitive abnormalities are among the most frequent and troublesome [10]. These problems usually produce low educational progress and underachievement throughout life [11]. Adults with epilepsy are also susceptible to cognitive degeneration [12]. Many adults with epilepsy frequently complain of memory disorders [13]. The most reported cognitive complaints in adults with epilepsy include mental slowness, memory impairment, attention deficits, persistent cognitive problems, and generating global intellectual deficits [14]. According to the theory of mind (ToM), patients with TLE have higher risk factors for cognitive dysfunctions [15–17]. This theory predicts a variety of neurocognitive processes in patients with epilepsy, such as cognitive and emotional dysfunctions [18]. Also, the model of perception in mirror neurons phenomenon and simulation theory [19] is helpful to recognize possible effects of epilepsy on cognitive dysfunctions. This

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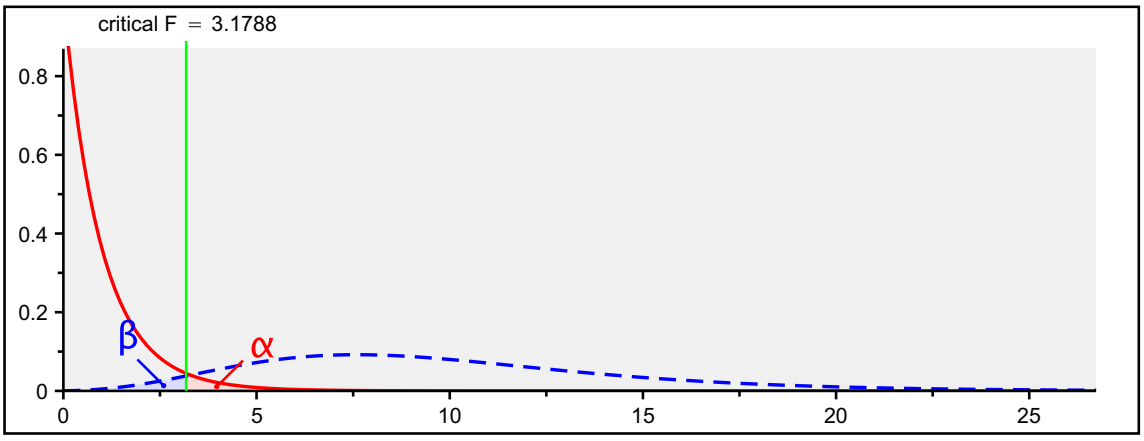

Fig. 1. Central and noncentral distributions of sample size.

theory assumes that disturbances in the imitation principle due to abnormal neural discharges relating to epilepsy can interfere in efficient cognitive functions.

Otherwise, it is known that migraine may be a form of progressive brain disorder that is related to a higher risk of cognitive impairment [20]. Patients with migraine, especially those followed at neurology clinics, show a superior threat of mild changes in several domains of cognitive functions, particularly in neuropsychological domains such as visual memory, verbal memory, information processing speed, attention, and executive functions [21]. A current study showed significant decline in neurocognitive functions of patients with migraine in verbal and visual memories; and reaction time [22]. Neuropsychological measurement suggests that there are some slight but significant changes in cognitive functions, both during, and between migraine episodes [23]. Overall, Torkamani et al. [24] showed that executive functions and episodic memory are mainly intact in patients with chronic migraine, and where deficits are observed, they are of little clinical relevance and do not affect the normal cognitive functions of the patients. According to the well-designed connectivity model in the frontoparietal network, patients with migraine have greater cognitive dysfunctions than healthy individuals [25,26]. Therefore, this study suggests that patients with OLE and patients with migraine would have higher cognitive and perceptual dysfunctions than the control group. The main hypothesis of this study is that patients with OLE and migraine, and healthy individuals would have significant different performances in cognitive and perceptual functions.

2. Method

2.1. Design and procedure

The study is a casual evaluation or quasi-experimental design with three groups [27]. These three groups include the following: outpatients with OLE, outpatients with migraine, and healthy individuals. Sample size is determined with regard to the research design with three groups and the robust statistic for one-way and multivariate analysis of covariance (MANCOVA) [28,29]. In the present study, participants are individuals who are relatively similar in some demographics (i.e., age, ethnicity, religion, and the number of years of education), but they only differ in their health status (i.e., OLE, migraine and normally healthy controls).

Patients with OLE and patients with migraine had their diagnoses and were confirmed by an accredited neurologist as having the clinical features of the aforesaid diseases, comorbid diseases, and differential diagnoses. All patients with OLE and patients with migraine in the clinical groups were recruited from several outpatient clinics. However, all patients in both clinical groups were selected by the purposive sampling method within a quasi-experimental design.

The purposive sampling often starts with a defined goal in mind, and the sample is thus selected to include people of concern and rule out those who do not fit the rationale [30,31]. The healthy individuals' diagnostic criteria were determined by a neurologist, and after his approval, they were assigned to the study. After reading and understanding the informed consent form, all participants had the option to ask any questions about the present study prior to signing the informed consent. After voluntary informed consent was obtained, participants completed a demographic questionnaire with BGT-II.

2.2. Participants

The sample consisted of 93 individuals (32 outpatients with OLE, 30 outpatients with migraine, and 31 healthy individuals) from Mashhad City, Khorasan-e-Razavi province, Iran. According to Wilson Van Voorhis and Morgan [29], this sample size is adequate for calculation of the statistical assumptions and comparisons between both the clinical and control groups. Sample size calculation using G*Power 3.1.9.2 justified on the basis of a predicted difference in the outcome among number of groups = 3, effect size = 0.56, α error probability = 0.05, and power (1-β error probability) = 0.95. This analysis showed that the total sample size = 54, critical $F = 3.178$, and actual power = 0.95 for this study (Fig. 1). The sample included 57 males and 36 females. The age mean and standard deviation for males and females were 25.33 (standard deviation [*SD*] = 8.24) and 26.08 years (*SD* = 7.76) respectively. The number of years of education in this sample was under five ($N = 5$), five ($N = 15$), six to twelve ($N = 17$), fourteen ($N = 33$), and sixteen ($N = 23$) in the total sample. All participants were Fars ethnic and Muslims.

Table 1
Normality assumptions for ANOVA.

BGT-II subscales	M	SD	Skewness	Kurtosis	Levene test of homogeneity of variances	
					F	*p*
Copy score	34.25	7.28	−0.74	−0.02	2.27	.06
Recall score	13.80	7.61	0.33	−0.10	1.719	.13
Motor score	10.83	1.87	−2.01	1.8	2.20	.07
Perceptual score	8.94	1.86	−2.19	1.9	2.14	.08

Note: $N = 93$, BGT-II = Bender–Gestalt II.

Table 2
Neurocognitive functioning in patients with occipital lobe epilepsy, patients with migraine, and healthy individuals.

BGT-II subscales	SS	df	MS	*F*	*p*
Copy score	1079.38	2	539.69	12.75	.0001
Recall score	1787.79	2	893.89	22.70	.0001
Motor score	80.18	2	40.09	14.76	.0001
Perceptual score	73.22	2	36.61	13.42	.0001

2.3. Instruments

A demographic questionnaire and Bender–Gestalt II were used for data gathering in this study. The demographic questionnaire included the participants' status, age, gender, and educational level questions.

2.3.1. Bender–Gestalt II (BGT-II) [32]

The Bender–Gestalt is a neurological, perceptual, developmental, personality, and visual–motor test that was developed by Lauretta Bender many years ago. This test originated into the Gestalt theory [33–35]. The Bender–Gestalt Test (BGT) [33] has provided insight into such problems as brain injury and neuropsychological pathology [36]. The BGT-II maintains Bender's original philosophy [33] and its validity and reliability confirmed using a normative sample of 4000 individuals for Copy and Recall procedures [32]. Bender–Gestalt-II is intended to diagnose neuropsychological functions in different situations [37–39]. The BGT-II measures visual–motor integration skills in individuals from 4 to 85 + years of age [40]. The BGT-II includes 13 designs for children below 8 years of age and 12 designs for individuals 8 years of age and older. The BGT-II comprises of four subscales: copy, recall, motor, and perceptual subscores. Copy component is a measure of visuomotor ability and perception and visuospatial and constructional skills. Recall component is a measure of visual memory and an index of short-term memory. The motor component is substantially equivalent to that of the copy task but relies on recalled internal representations of the items. The perceptual component shows a mixture of picture completion, picture arrangement, figures design, and assembly of various details in a given figure. The BGT-II discriminant and concurrent validity were confirmed for an Iranian population. The BGT-II also has good reliability [41].

2.4. Statistical analysis

To evaluate the major hypotheses in the present study four analyses of variances (ANOVAs) by group status (as fixed independent variable) and BGT-II (i.e., four subscales) as dependent variables were calculated in this sample. In addition, a MANCOVA by group status, gender, and the levels of education (as fixed independent variables) and the BGT-II (i.e., four subscales) as dependent variables were calculated in this sample.

3. Results

To examine the main hypothesis, all basic normality assumptions were computed, which shows a normal distribution for running of ANOVAs in this study (Table 1). This analysis showed the role of group status, $F(2, 92) = 12.75$; $p < .0001$; in copy, $F(2, 92) = 22.70$; $p < .0001$; recall, $F(2, 92) = 14.76$; $p < .0001$; motor, and $F(2, 92) = 36.61$; $p < .0001$; perceptual subscales of BGT-II in this sample (Table 2). Tests of between-subjects' effects using the Duncan posthoc test indicated significant differences among patients with OLE, patients with migraine, and normal individuals in the control group (Table 3).

To compute the MANCOVA, Box's test of equality of covariance matrices showed a normal distribution of dependent variables as the basic assumption of MANCOVA (Box's M = 88.61, $F = 1.18$, p ˃ .17). Again, this analysis indicated the role of group status (*Wilks'* $k = 0.750$; $F(8, 134) = 2.58$; $p < .01$;) in all dependent variables. However, this analysis rejected the role of gender (*Wilks'* $k = 0.846$; $F(4, 67) = 1.13$; $p < .17$;) and the levels of education (*Wilks'* $k = 0.157$; $F(16, 205) = 0.843$;) in all of the independent variables.

4. Discussion

The results for the main hypothesis demonstrated significant group differences for copy, recall, motor, and perceptual subscales of BGT-II in this sample. There were no significant differences for gender and the levels of education in all the dependent variables in the present study. The Duncan test of posthoc comparisons for group differences showed that both patients with OLE and patients with migraine have significantly lower scores than normal individuals in copy, recall, motor, and perceptual subscales of BGT-II. In addition, patients with OLE had significantly lower scores in copy, recall, motor, and perceptual subscales of BGT-II in comparison to patients with migraine. These results are consistent with the predictions of Gestalt psychology that postulates the perceptual experience as a holistic pattern of stimuli, which is the result of interaction between innate and biological endowment and the mind [33–35,38,42]. The entire integrated status of a human will thus affect performance on the BGT-II. Any individual will recognize and rearrange BGT-II figures according to the integrative state of his/her internal world, which will vary in different levels of neuropsychological impairments. As Pascal and Suttell [43] noted already, a good performance in BGT is dependent on well-organized sensory perception, interpretation, and motor reproduction between internal world and the external stimuli.

These results are congruent with theoretical predictions of cognitive and perceptual dysfunctions in patients with epilepsy and patients with migraine [15–19,25,26]. In agreement with the predictions of these theories ;, the present findings show the higher cognitive and perceptual dysfunctions in both patients with OLE and patients with migraine in comparison to the healthy controls. This study suggests that abnormal neurophysiological activity of the brain in patients with OLE and patients with migraine will result in cognitive and perceptual dysfunctions. Moreover, the present results are congruent with previous literature that supported many shared psychological features in patients with epilepsy and patients with migraine diseases [2–4,44]. Thereby, these results verify the resembled neuropsychological and cognitive–perceptual functions in patients with epilepsy and patients with migraine. The present results align and support previous findings in the literature that show more neuropsychological dysfunctions in patients with epilepsy than those with migraine [5–10,12–14,20,21]. Similarly,

Table 3
Mean and standard deviation of BGT-II subscales in patients with occipital lobe epilepsy, patients with migraine, and healthy individuals.

BGT-II subscales	Patients with occipital lobe epilepsy ($N = 32$)		Patients with migraine ($N = 30$)		Healthy individuals ($N = 31$)	
	M	SD	M	SD	M	SD
Copy score	30.31	6.73	34.00	7.83	38.58	4.55
Recall score	8.75	5.86	13.43	6.21	19.38	6.72
Motor score	8.40	2.33	8.10	1.49	11.83	0.58
Perceptual score	6.75	2.56	7.33	1.09	9.80	0.47

the higher incidence of cognitive and perceptual dysfunctions in patients with OLE and patients with migraine are consistent with the earlier findings of neuropsychological dysfunctions, which thereby affirm the biological underpinning of cognitive deficiencies in patients with epilepsy [5–10,12–14].

In conclusion, this study adds to the current literature in the field of cognitive and perceptual functions about chronic neurological diseases, by demonstrating the incidence of significantly lower levels of copy, recall, and motor performances and higher perceptual dysfunctions in patients with OLE and patients with migraine in comparison with those of normal individuals. Clinicians and mental health professionals may use BGT-II for screening and treatment goals in patients with OLE and patients with migraine. However, the present study has limitations because it has only used the BGT-II measure in patients with OLE and patients with migraine. Also, the present sample was not reflecting the epidemiology of migraine by sex and the educational level of the Iranian population. Further studies are essential to recognize how the type and severity of different neurophysiological abnormalities can impact the quantity and quality of cognitive–perceptual dysfunctions in BGT-II among patients with OLE and patients with migraine. It would be expected that further investigations to evaluate cognitive–perceptual dysfunctions in patients with OLE and patients with migraine will require BGT-II and other concurrent and more sophisticated measures.

Declaration of Competing Interest

None.

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