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Title: Brain Wave Patterns in Patients With Chronic Low Back Pain: A Case-Control Study

Running Title: Brain wave patterns and chronic LBP

Authors: Maryam Sadat Larie¹, Fateme Esfandiarpour^{1,2*}, Forough Riahi³, Mohamad Parnianpour⁴

1. Department of Physiotherapy, Musculoskeletal Rehabilitation Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

2. Department of Family Medicine, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Canada.

3. Department of, Psychiatry, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

4. Department of Mechanical Engineering, Sharif University of Technology, Tehran, Iran.

***Corresponding Author:**

Fateme Esfandiarpour, PhD. Address: Department of Physical Therapy, School of Rehabilitation Sciences, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran. E-mail: fateme@ualberta.ca

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Abstract

Introduction: Research evidence indicates that maladaptive reorganization of the brain plays a critical role in amplifying pain experiences and pain chronification; there is, however, no clear evidence of change in brain wave activity among patients with chronic low back pain (CLBP). The objective of this study was to assess brain wave activity in patients with CLBP, compared to healthy controls.

Methods: Twenty-five patients with CLBP and twenty-four healthy controls participated in the study. A quantitative Electroencephalography device was used to assess brain wave activity in eyes-open and eyes-closed conditions (EO and EC). The regional absolute and relative power of brain waves were compared between the groups.

Results: Our results showed significant increases in the absolute power of theta ($F=5.905$, $p=0.019$), alpha ($F=5.404$, $p=0.024$) waves in patients with CLBP compared to healthy subjects in both EC and EO condition. Patients with CLBP showed a reduced delta absolute power in the frontal region ($F=5.852$, $p=0.019$) and augmented delta absolute power in the central region ($F=5.597$, $p=0.022$) in the EO condition. An increased delta absolute power was observed in the frontal ($F=7.563$, $p=0.008$), central ($F=10.430$, $p=0.002$) and parietal ($F=4.596$, $p=0.037$) regions in patients with CLBP compared to the healthy subjects in the EC condition. In the EC condition, significant increases in theta relative power ($F=4.680$, $p=0.036$) in the parietal region was also found in patients with CLBP.

Conclusion: Increased absolute power of brain waves in people with CLBP may indicate cortical overactivity and changes in pain processing mechanism in these patients.

Keywords: Brain Wave, Chronic pains, Low Back Pains, Musculoskeletal pains, Neuroplasticity

Highlights

- CLBP increases the alpha, theta and delta power in the brain.
- CLBP is associated with an increase in brain wave activity of the frontal, central and parietal regions.
- Our findings suggest altered central pain processing in CLBP.

Plain Language Summary

Traditional diagnosis and treatment of CLBP are mainly focused on peripheral pathology. But, the modern neuroscience approach to pain highlights the role of cortical plasticity in chronic musculoskeletal pain. In this regard, several studies found structural and functional changes in the brain in patients with chronic pain. Detailed knowledge about cortical changes in CLBP can improve our understanding of mechanisms involved in CLBP open a new window to better treatment of LBP. This study investigated brain wave activity in patients with CLBP in comparison to healthy individuals. Our findings suggest increased brain activity in various parts of the brain in patients with chronic LBP. This finding indicates that CLBP treatment should focus on both peripheral and cortical factors rather than local tissue damage.

1. Introduction

Low back pain (LBP) is the most common chronic pain, affecting 70% – 85% of the adult population at some point in life (Becker et al., 2010). About 10%-30% of people with LBP develop chronic pain leading to early retirement and high healthcare costs (Becker et al., 2010; Johannes, Le, Zhou, 2010). Treatment of CLBP comprises rest, pharmacological treatment, and physiotherapy, which are usually effective only in the short-term (Qaseem, Wilt, McLean, 2017; Saragiotto et al., 2016). The increasing worldwide prevalence of CLBP and inadequate long-term outcomes of current treatment urged a reassessment of the traditional approaches for CLBP treatment.

Traditional diagnosis and treatment of CLBP are mainly focused on peripheral pathology and mechanical abnormalities (Ferreira, Ferreira, Maher, 2006); however, the modern neuroscience approach to pain highlights the role of cortical plasticity in pain chronification and amplification (Fregni, Freedman, Pascual-Leone, 2007). From a neuroscience perspective, maladaptive reorganization of the brain neural networks plays a critical role in chronic pain conditions (Apkarian, 2011; De Vries et al., 2013). In this regard, neuroimaging studies revealed structural and functional changes of the brain in chronic musculoskeletal pain conditions, supporting the neuroscience approach to chronic pain (Kregel et al., 2015). For instance, Changes in the brain's EEG activity were also found in individuals with neuropathic pain and fibromyalgia (González Roldán, Muñoz, Cifre, 2013; Sarnthein, Stern, Aufenberg, 2006). Similarly, researchers reported changes the brain morphology and function in patients with CLBP as compared to pain-free controls (Apkarian et al., 2004; Giesecke et al., 2004; Schouppe, Van Oosterwijck, Danneels, 2019). Employing Magnetic Resonance Imaging (MRI), Magnetoencephalography (MEG), and Transcranial Magnetic Stimulation (TMS), previous studies on patients with CLBP have evidenced a significant reduction in gray matter density in prefrontal and thalamic regions (Apkarian et al. 2004) as well as alterations in primary somatosensory (Kregel et al. 2015) and motor cortex representation of the back (Tsao, Galea, Hodges, 2008).

Previous studies investigated cortical changes in people with chronic pain, using TMS, MEG, Functional MRI, Positron Emission Tomography (PET), and Electroencephalography (EEG) (Apkarian, Bushnell, Treede, 2005; Schabrun, Jones, Cancino, 2014). Recently, quantitative Electroencephalography (qEEG) has also been considered by researchers, as it provides reliable information about brain function during rest, sensory stimulation, and cognitive tasks. In addition, this method is safe, inexpensive, and non-invasive, making it a valuable apparatus for clinical practice and research applications (De Vries et al. 2013; Spronk, Arns, Barnett, 2011). Nevertheless, despite the wide applicability of qEEG for evaluation of cortical changes in chronic musculoskeletal conditions, relatively little knowledge exists on brain wave activity changes in CLBP patients.

Brain waves are patterns of rhythmic or repetitive neural activity produced by synchronized oscillations of group of neurons that communicate with each other (Kropotov, 2010). According to frequency, brain waves are classified into delta, theta, alpha, beta and gamma waves (Jatoi & Kamel, 2018). Delta wave (1–4 Hz) is associated with deep sleep and a very relaxed state of mind (Guyton, 2011). A study showed delta activity is related to sustained pain and homeostatic processes (Knyazev, 2012). Theta wave (4–8 Hz) is involved in

emotions, focused concentration, and mental tasks (Tatum, Hausain, Banbadis, 2008). A review showed an increased theta activity in chronic pain patients (dos Santos Pinheiro et al., 2016). Alpha wave (8–12 Hz) represents a state of wakeful rest (Jatoi & Kamel, 2018). Research evidence shows that alpha wave is involved in basic cognitive processes and momentary memory storage, and inhibitory processes (Kropotov 2010, Klimesch, Sauseng, Hanslmayr, 2007). Moreover, a review study showed increased alpha power in chronic pain patients (dos Santos Pinheiro et al., 2016). Beta wave (12-30 Hz) is related with active thinking, concentration, and computation (Jatoi & Kamel, 2018). Previous studies reported altered the beta power in patients with chronic pain (Ploner, Sorg, Gross, 2017).

To our knowledge, quantitative brain activity in chronic back pain has, thus far, been investigated by Schmidt et al., who found no significant difference in brain activity between people with chronic back pain and their healthy counterparts. However, investigation of brain activity in Schmidt et al.'s study was limited to eyes-closed condition and parieto-occipital region (Schmidt et al., 2012). Therefore, we aimed to compare brain waves in people with CLBP and healthy subjects across three cortical regions in both eyes-open and eyes-closed conditions. Prior studies suggest that chronic pain conditions are associated with increased brain wave activity (dos Santos Pinheiro et al., 2016, Olesen et al., 2011). Thus, we hypothesized that CLBP is associated with increases in brain wave activity in comparison to healthy individuals.

2. Materials and Methods

This case-control study involved twenty-five participants (37.12 ± 5.86 years, 27.15 ± 2.7 kg/m²), with a diagnosis of CLBP, and twenty-four healthy participants (34.45 ± 4.01 years, 28.64 ± 3.69 kg/m²), with no history of chronic pain. The number of participants was calculated using our preliminary data on 10 subjects in each group ($\beta=0.20$ and $\alpha=0.05$) for between group differences in the regional theta power as the most-noticed brain wave abnormality in patients with chronic pain (Ploner, Sorg, Gross, 2017). Based on this analysis, a minimum of nineteen participants per each group was required.

Statement of Ethics

This study was approved by the Ethics Committee of the university (1396.713). All participants read and signed informed consent prior to testing.

For CLBP patients, the inclusion criteria were a history of persistent LBP for more than six months or recurring LBP for at least three incidents in the last year requiring activity limitation or medical intervention (Gombatto, Norton, Sahrman, 2013); age of 30-50 years; and an average pain severity of greater than 30 on a 0-100 mm visual analogue scale (VAS) in the last week. The healthy subjects were included if they had no history of chronic pain and age 30-50 years.

The exclusion criteria were the use of medications affecting brain wave activity, alcoholism and addiction, a history of inflammatory joint disease, systemic metabolic disorder, and/or neurological disorders, such as headache, seizure, polyneuropathy and psychological disorders, spondylolysis and spondylolisthesis, spinal surgery, trauma to low back, disc herniation based on an MRI report, pregnancy and physiotherapy in the last six months.

Data Collection

In this study, the regional brain wave activity in both eyes-open and -closed conditions (EO, EC), pain intensity, and functional ability were assessed.

Brain wave activity

The absolute and relative power of brain wave activity was recorded by quantitative Electroencephalograph (qEEG: Mitsar-EEG-201, Mitsar Co. Ltd, Saint-Petersburg, Russia) in both EO, EC conditions. QEEG is a method based on quantification of brain wave amplitudes and designed to process EEG signals (Kropotov, 2010). The absolute power index measures the energy of the electrodes in a certain region of the brain in different frequency bands in microvolt sq (Cunha et al., 2004). Relative power is power in each band, relative to the total frequency bands (Kropotov, 2010).

For the qEEG recording, the participants sat on a reclining chair in a semi-dark room, with 19 electrodes were placed on their scalp, according to the international 10-20 system (Jasper, 1958). The reference electrodes were placed on the earlobes. The maximum electrode impedance was 5 K ohms. The low and high cut filters were set at 0.53 and 30 Hz respectively, and the notch filter was set at 45-55 Hz. The sampling rate was 500 per second. Participants were asked to remain still during the recordings.

All EEG recordings were obtained between 8 am to 12 pm to avoid sleepiness. In order to prevent caffeine-induced theta decrease, the participants refrained from caffeinated drinks on the day of the qEEG recording (Landolt et al., 2004). In the EO condition, the subjects were requested to keep eyes open and maintain gaze on a fixation mark on the wall. In the EC condition, participants were instructed to close their eyes but not sleep.

QEEG frequency bands include delta (1.0-4.0 Hz), theta (4.0-8.0 Hz), alpha (8.0-12.0 Hz), and beta (12.0-25.0 Hz). The EEG data collection was done for five minutes per EO and EC conditions. The raw file of the EEG data was then sent to the Neuroguide software system for analysis and removal of artifacts (Kropotov, 2010). The EEG recordings were also visually inspected for artifact deletion. Artifact deletion was done based on eye movements, muscle movements and drowsiness using the artifact rejection toolbox of the Neuroguide system. Next, the artifact-free epochs were chosen for spectral analysis by the same software system, which uses spectral analysis to decompose the EEG pattern into simple components (sinusoidal waves) using Fast Fourier Transformation (FFT): a method for converting brain wave data from the time domain to the frequency domain, so as to create absolute and relative power indices (Kropotov, 2010). The values obtained from FFT absolute and relative power were entered into the SPSS statistical software for analysis. The average absolute and relative power of each frequency band was calculated for brain regions relevant to pain processing, i.e., the frontal (Fp₁, Fp₂, F₃, F₄, F₇, F₈, F_z), central (C₃, C₄, C_z) and parietal (P₃, P₄, P_z) regions.

Pain intensity

Pain intensity was assessed with a 100 mm (visual analog scale) VAS, with 0 for a painless state and 100 for the worst imaginable pain. The participants were requested to rate their mean pain intensity in the week prior (Carlsson, 1983).

Oswestry Disability Index

The Oswestry Disability Index questionnaire was used to rate participants' functional ability (Mousavi, Parnianpour, Mehdian, 2006). This questionnaire consists of 10 sections of six items with a range score of 0-50, in percentages. The disability score of 0-20%

indicates minimal disability, 21-40% moderate disability, 41-60% severe disability, 61-80% crippled, and 81-100% bed-ridden, with worsening symptoms (Fairbank & Pynsent, 2000).

Statistical analysis

First, the normality of data was assessed by the K-S test. Mixed ANOVA tests (3 regions $\times$ 2 condition $\times$ groups) were used to compare differences in the regional absolute and relative power between the groups in both conditions. Univariate test was used to contrast differences in the regional absolute and relative power of the brain waves between the groups when the main effect of group was significant. The level of significance was set at $P < 0.05$ and the confidence interval was considered as 95% CI for all analysis. The data were analyzed in SPSS v.22 (IBM, Chicago, IL).

3. Results

There were no significant differences between the groups for demographic characteristics of age and BMI ($p > 0.05$). Participants with CLBP had a mean pain intensity of 48 ± 11.45 and a disability score of $27.52 \pm 9.4/100$ based on VAS and the Oswestry Disability Index respectively. The participants in the control group had an Oswestry score of zero.

The mean and SE of the absolute and relative power of brain waves for healthy controls and CLBP patients in both conditions are reported in Table 1 and 2. Tables 3 and 4 showed the absolute and relative power of brain waves in the frontal, central and parietal regions in both eye open and eye closed conditions. Three-way interaction was not significant for the absolute power of theta, alpha and beta waves and the relative power of the delta and alpha waves. In other cases, the three-way interaction was significant.

There were significant increases in the regional absolute power of theta ($F = 5.905$, $p = 0.019$), (mean difference = 6.86, 95% CI (1.18, 12.55) and alpha ($F = 5.404$, $p = 0.024$), (mean difference = 13.97, 95% CI (1.82, 26.07) waves in patients with CLBP in both condition (Table 3). In the EO condition, patients with CLBP showed a reduced delta absolute power in the frontal region ($F = 5.852$, $p = 0.019$) and augmented delta absolute power in the central region ($F = 5.597$, $p = 0.022$) (Table 5). In the EC condition, significant increases were found in the regional absolute power of delta in the frontal ($F = 7.563$, $p = 0.008$), central ($F = 10.430$, $p = 0.002$) and parietal ($F = 4.596$, $p = 0.037$) regions in patients with CLBP compared to the healthy subjects (Table 5).

Significant increases in the relative power of theta wave ($F = 4.680$, $p = 0.036$) in the parietal region was also found in patients with CLBP in the EC condition (Table 6). We detected no significant differences between the groups in the regional absolute and relative power of the beta wave ($p > 0.05$) (Tables 3 and 6).

Table 1. Mean (SE) of the absolute power of brain waves for CLBP patients and healthy controls in both conditions

Eye condition	Brain waves	CLBP group			Healthy control		
Eyes		Frontal	Central	Parietal	Frontal	Central	Parietal
open	Delta	16.95(1.22)	17.91(1.15)	17.3(1.29)	21.18(1.24)	14.02(1.17)	13.63(1.32)
	Theta	10.98(1.76)	14.13(2.48)	13.34(2.68)	7.29(0.49)	7.96(0.59)	7.03(0.60)
	Alpha	10.08(1.91)	20.03(4.46)	34.17(8.26)	5.57(0.54)	8.69(0.96)	12.88(2.34)
	Beta	7.65(0.84)	9.84(1.68)	11.10(2.01)	5.94(0.39)	6.70(0.49)	7.16(0.55)
Closed	Delta	17.28(0.81)	16.49(0.86)	16.09(1.08)	14.06(0.83)	12.5(0.88)	12.77(1.10)
	Theta	12.89(1.79)	18.98(3.77)	21.06(5.31)	8.41(0.54)	9.61(0.73)	9.87(1.05)
	Alpha	22.92(3.80)	36.45(6.45)	66.88(14.52)	15.04(1.67)	21.23(2.45)	43.26(7.01)
	Beta	7.54(0.83)	12.25(2.07)	15.54(2.78)	6.45(0.43)	9.14(0.75)	12.35(1.10)

CLBP: Chronic Low Back Pain; SE: Standard Error

Table 2. Mean (SE) of the relative power of brain waves for CLBP patients and healthy controls in both conditions

Eye condition	Brain waves	CLBP group			Healthy control		
Eyes open		Frontal	Central	Parietal	Frontal	Central	Parietal
	Delta	41.81(2.22)	34.22(2.75)	30.35(2.95)	44.60(1.65)	36.34(1.44)	34.06(1.66)
	Theta	19.75(1.04)	21.18(1.30)	17.09(1.52)	17.91(0.70)	19.43(0.67)	16.61(0.41)
	Alpha	17.06(1.63)	25.19(2.50)	34.30(3.46)	13.99(0.98)	20.86(1.42)	26.72(2.31)
	Beta	15.14(0.85)	15.77(1.18)	15.40(1.15)	15.12(0.75)	17.03(0.94)	17.93(0.93)
Eyes Closed	Delta	32.17(2.39)	25.32(2.28)	21.08(2.38)	32.31(1.67)	24.68(1.50)	19.10(1.60)
	Theta	19.62(1.31)	21.32(2.07)	18.52(2.52)	17.75(0.72)	17.80(0.92)	12.77(0.68)
	Alpha	32.67(2.64)	40.30(4.01)	53.28(8.54)	30.40(2.23)	35.87(2.32)	48.35(2.79)
	Beta	12.84(0.87)	15.51(1.45)	15.98(1.72)	13.93(0.65)	16.96(0.76)	16.12(1.01)

CLBP: Chronic Low Back Pain; SE: Standard Error

Table 3: Analysis of variance for absolute power of brain waves at brain regions of frontal, central and parietal in eye-closed and eye-open conditions for healthy controls and patients with CLBP

		Theta (uV ²)	Alpha (uV ²)	Beta (uV ²)
Main effect				
Group	F	5.905	5.404	2.336
	p	0.019*	0.024*	0.133
Eye Condition	F	7.549	38.132	40.162
	p	0.008*	<0.0001*	<0.0001*
Region	F	5.035	22.050	22.624
	p	0.008*	<0.0001*	<0.0001*
Interaction				
eye condition*group	p	0.231	0.267	0.554
region*group	p	0.066	0.129	0.260

Table 4: Analysis of variance for relative power of brain waves at brain regions of frontal, central and parietal in eye-closed and eye-open conditions for healthy controls and patients with CLBP

		Delta (%)	Alpha (%)
Main effect			
Group	F	0.144	1.332
	p	0.706	0.254
Eye Condition	F	47.000	38.778
	p	<0.0001*	<0.0001*
Region	F	129.597	52.380
	P	<0.0001*	<0.0001*
Interaction			
eye	p	0.043*	0.782
condition*group			
region*group	p	0.870	0.569

Table 5. Univariate analysis of delta absolute power between patients with CLBP and healthy individuals in both conditions

Absolute power (μV^2)	F	P	Mean difference (95 % CI)
Eyes Open			
Frontal	5.852	0.019*	-4.23 (-7.74, -0.71)
Central	5.597	0.022*	3.88 (0.58, 7.19)
Parietal	3.940	0.053	3.67 (-0.04, 7.39)
Eyes Closed			
Frontal	7.563	0.008*	3.21 (8.64, 5.57)
Central	10.430	0.002*	3.99 (1.50, 6.48)
Parietal	4.596	0.037*	3.31 (0.20, 6.43)
CLBP: Chronic Low Back Pain; CI: confidence interval; *Significant at $p < 0.05$			

Table 6. Univariate analysis of theta and beta relative power between patients with CLBP and healthy individuals in both conditions

Brain Wave	relative power (%)	F	P	Mean difference (95 % CI)
Theta	Eyes Open			
	Frontal	2.078	0.156	1.83(-0.72,4.40)
	Central	1.369	0.248	1.74 (-1.25, 4.74)
	Parietal	0.088	0.768	0.47 (-2.75, 3.71)
	Eyes Closed			
	Frontal	1.515	0.225	1.87(-1.18, 4.93)
	Central	2.322	0.134	3.51(-1.12, 8.15)
	Parietal	4.680	0.036*	5.75 (-0.40, 11.10)
Beta	Eyes Open			
	Frontal	0.000	0.987	0.02(-2.29, 2.32)
	Central	0.684	0.412	-1.25(-4.31, 1.80)
	Parietal	2.887	0.096	-2.53(-5.54, 0.46)
	Eyes Closed			
	Frontal	0.972	0.329	-1.08(-3.30, 1.13)
	Central	0.753	0.390	-1.44(-4.80,1.90)
	Parietal	0.005	0.941	-0.14(-4.21, 3.91)

CLBP: Chronic Low Back Pain; CI: confidence interval; *Significant at $p < 0.05$

4. Discussion

In this study, we compared regional brain wave activity in CLBP patients with healthy subjects. Our findings showed significant increases in the regional absolute power of brain waves in patients with CLBP compared to their healthy counterparts. Cortical overactivity in patients with CLBP may indicate altered pain processing mechanisms in CLBP when compared to healthy individuals.

This study detected increased regional brain activity of the delta and theta frequency bands in patients with CLBP. These findings suggest increased brain oscillations of the regions tested, indicating cortical neuroplastic changes in patients with chronic LBP. In line with our findings, an extensive shift in brain organization and morphology in patients with CLBP was observed in neuroimaging studies using TMS, MRI and voxel-based morphometry (VBM) (Apkarian et al., 2004; Tsao et al., 2008). For instance, previous studies found changes in trunk muscle representation at the motor cortex (Tsao et al., 2008), reduction in neocortical gray matter volume (Apkarian et al., 2004) in patients with CLBP. Extensive brain activity changes in people with CLBP can be explained based on the neuromatrix theory (Melzack, 1999). According to the neuromatrix theory, pain is produced by specific "neurosignature" patterns of nerve impulses, generated by a complex neuronal network, distributed in different regions of the brain (SAAB, 2014). Contrary to our finding, Schmidt et al. investigating brain activity in the parieto-occipital region found no significant difference between people with CLBP and their healthy counterparts in the brain activity, averaged across the parieto-occipital sites. However, limitations in the study by Schmidt et al. such as the use of medications by about 50% of participating subjects, investigation of brain activity only in the parieto-occipital region and eye-closed condition make it hard to compare our findings to theirs (Schmidt et al., 2012).

Our findings showed significant overactivity of the delta wave in people with CLBP when compared to healthy controls in the EC condition. However, we found reduced delta wave activity in the frontal region in the EO condition. An overactivity of delta wave was also observed in patients with chronic pancreatitis and migraine (Olesen et al., 2011; Bjørk et al., 2009). Similar to our findings, Hargrove et al. found lower delta absolute power in the frontal region in patients with fibromyalgia (Hargrove et al., 2010).

We also found significant increases in the regional absolute power of theta in patients with CLBP in both conditions. In the EC condition, there was a significant increase in the relative power of theta wave in patients with CLBP. Increased theta activity is a common finding in patients with chronic pain (dos Santos Pinheiro et al., 2016). For instance, theta wave overactivity was found in patients with fibromyalgia (González Roldán et al., 2013; Fallon, Chiu, Nurmikko, 2018) and neurogenic pain (Sarnthein et al., 2006; Stern, Jeanmonod, Sarnthein, 2006). Animal studies suggest that theta waves in the parietal region can be predictive of pain (LeBlanc, Lii, Silverman, 2014). Increased theta activity in chronic pain was explained by TCD theory (Llinás, Urbano, Leznik, 2005).

Thalamocortical Dysrhythmia refers to a state in which thalamic neurons change from high-threshold tonic firing to low-threshold calcium spike bursts in the theta–delta range caused by the suppression of excitatory inputs onto thalamic cells (Jahnsen & Llinas, 1982; Jahnsen &

Llinas 1984 a, b; Steriade, Datta, Pare, 1990; Jeanmonod, Magnin, Morel, 1996). In this regard, studies showed the behavior of the thalamus neurons alters in chronic pain disorders, which in turn disrupts the normal thalamocortical rhythm (De Vries et al., 2013; Pascoal Faria, Yalcin, Fregni, 2015). This disruption results in alterations in cortical processing in the frequency domain, including exceeding of theta power (De Vries et al., 2013).

Our results showed an increased alpha power in the regional absolute power in patients with CLBP compared to the healthy controls in both conditions. Consistent with our findings, several studies reported increased alpha activity in patients with chronic pain conditions such as chronic jaw pain (Wang et al., 2019), trigeminal neuralgia (Wang et al., 2014) and chronic pancreatitis (Drewes et al., 2008). Increased alpha activity indicates lower cortical excitability and a higher perceptual threshold (Foxe & Synder, 2011). Increased alpha activity in patients with chronic pain may correspond with functional suppression of attention (Wang et al., 2019). Research findings also showed that higher alpha power is associated with the lower subjective perception of pain (Babiloni et al., 2016).

Brain waves including delta, theta, and alpha are related to cognitive performance (Klimesch, 1999; Prichep et al., 1994). Cognitive dysfunction is commonly observed in chronic pain patients (Berryman et al., 2013). For instance, a review study showed increased theta and alpha power are associated with reduction in the amplitude of evoked potentials in cognitive tasks in patients with chronic pain (dos Santos Pinheiro et al., 2016). Also, another study reported increased theta power is related to reduced cognitive efficiency in patients with fibromyalgia (Donaldson, Mueller, Donaldson, 2003). So, it is suggested that future studies assess correlation between brain wave pattern and cognitive function in patients with chronic low back pain.

Limitations

The present study had several limitations. In this study, we focused on the absolute and relative power indices between the groups. We did not investigate other indices of qEEG, such as coherence, power ratio, peak frequency, phase lag, and amplitude asymmetry. We did not use source localization techniques, such as LORETA to detect the exact region of the brain involved. Thus, future studies are required to examine brain source localization along with qEEG analysis.

Conclusion

Our findings indicate a widespread overactivity of brain waves in patients with CLBP compared to their healthy counterparts. These alterations in brain wave activity may play a principal role in chronification and recurrence of pain in people with LBP. Cortical changes associated with CLBP present a new target for treating CLBP. Future studies aiming at the effects of cortical stimulation on central neuroplastic changes in CLBP will further our understanding of the cortical changes associated with LBP and possible treatment strategies to tackle this clinical dilemma.

Ethical Considerations

All ethical principles were considered in this article. This study was approved by the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (IR.AJUMS.REC.1396.713) and was registered in Iranian Registry for Clinical Trial (IRCT20140705018362N3).

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Author Contributions

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Conflict of interest

The authors declare no conflict of interest related to this manuscript.

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