Cerebral Representation of Splint Therapy in Bruxism Patients; An Functional Magnetic Resonance Imaging Experiment

Bruksizm Hastalarında Splint Tedavisinin Serebral Gösterimi; Bir Fonksiyonel Manyetik Rezonans Görüntüleme Deneyi

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ABSTRACT

Objective: The aim of this study was to investigate the effects of the novel Nociceptive Trigeminal Inhibition Tension Suppression System (NTI-tss) splint therapy in bruxism patients based on the view that the etiology is derived from central nerveous system. It was also aimed to evaluate the differences in cortical activity between bruxism patients and healthy subjects during the clenching function.

Material and Methods: A total of 30 volunteers (15 bruxist and 15 healthy) attending the KırıkkaleUniversity Faculty of Dentistry for various dental reasons were included in the study. For the diagnosis of bruxism, the criteria determined by an international consensus in 2013 were used. Brain activity was examined by functional magnetic resonance imaging during tooth clenching and rest tasks for each participant with the BrainVoyager QX 2.8 statistical data analysis program. This protocol was approved by the ethics committee of the medical faculty of Kırıkkale University, Turkey (02/04).

Results: When the average rate of brain activity in healthy subjects and bruxism patients was compared, hypoactivation was observed during the clenching task in the inferior parietal lobe (Brodmann 39 ve 40) and dorsal posterior cingulate area (Brodmann 31). The group analysis between pre- and post- treatment groups revealed one activation cluster in the post-treatment group that may indicate increased activation in the mentioned cortical areas after 11 nights of NTI-tss splint therapy. When control group compared with post- treatment group no differences observed.

Conclusion: With the aid of a neuroimaging technique, the decrease in activation in certain areas of the cerebral cortex in patients with bruxism. Our findings indicate that wearing the NTI-tss splint for 11 nights increased BOLD in the mentioned cortical area. These results might improve the understanding and physiological handling of sleep bruxism.

Key Words: Bruxism, fMRI, Magnetic resonance imaging, Mastication, Occlusal splint

ÖZ

Amaç: Çalışmanın amacı bruksimin etiyolojisinin santral sinir sistemi kaynaklı olduğu görüşünden yola çıkarak Nociceptive Trigeminal Inhibition Tension Suppression System (NTI-tss) okluzal splint tedavisinin beyin merkezlerinde meydana getireceği olası değişiklikleri gözlemlemektir. Ayrıca bruksizmi olan hastalar ve sağlıklı bireyler arasında diş sıkma fonksiyonu sırasında kortekste oluşan aktivite farklarının değerlendirilmesi amaçlanmıştır.

Gereç ve Yöntemler: Çalışmaya Kırıkkale Üniversitesi Diş Hekimliği Fakültesi Ağız Diş ve Çene Radyolojisi Anabilim Dalına çeşitli şikayetlerle başvuran 15 bruksizmi olan ve 15 bruksizmi olmayan (kontrol grubu) toplam 30 birey katılmıştır. Bruksizm varlığının belirlenmesinde 2013 yılında uluslar arası otörler tarafından düzenlenen kriterler kullanılmıştır. Her katılımcı için diş sıkma ve dinlenme görevleri sırasında fonksiyonel manyetik rezonans görüntüleme ile beyin aktivitesi incelendi. Data analizi için BrainVoyager QX 2.8 programı kullanılmıştır. Bu proje Kırıkkale Üniversitesi Etik kurulu (Karar no: 02/04) tarafından onaylanmıştır.

Bulgular: Sağlıklı bireyler ile bruksizmi olanlar arası diş sıkma görevi sırasında beyinde meydana gelen aktivasyon ortalaması karşılaştırıldığında bruksizm grubunda inferior parietal lob (Brodmann 39 ve 40) ve dorsal posterior cingulate area (Brodmann 31) bölgelerinde hipoaktivasyon gözlenmiştir.

Splint kullanımı öncesi ve sonrası gruplar analiz edildiğinde 11 günlük splint kullanımı sonrasında aktivitede artış kümesi izlendi; kontrol grubu ile bruksizm grubu arasında anlamlı bir fark bulunmamıştır.

Sonuç: Bir nörogörüntüleme tekniği olan fMRI tekniği yardımı ile bruksizm hastalarında serebral korteksin belirli bölgelerinde aktivasyonda azalma izlenmiştir. 11 günlük splint kullanımının bu bölgelerde BOLD sinyalini artırdığı saptandı. Bu çalışmanın bulgularının bruksizmin etiyolojisi ve etkin tedavisinin netleşmesine yardım edeceğini düşünmekteyiz.

Anahtar Sözcükler: Bruksizm, fMRI, Manyetik rezonans görüntüleme, Çiğneme, Okluzal splint

INTRODUCTION

Bruxism is known as a non-functional behavior of the masticatory muscles that occurs nocturnally and diurnally (1). Many practitioners believe that bruxism is caused by psychological stress (2, 3). Such statements are not strongly evidence-based. The majority of sleep bruxism patients do not present with any concomitant medical or psychiatric conditions (idiopathic or primary bruxism), while daytime bruxism patients have been reported to depict multisystem atrophy, cervical dystonia, basal ganglia infarction or use and deprivation of some drugs (secondary bruxism) (4, 5). There are various explanations on the etiology and pathophysiology of this disorder but none of them have been scientifically confirmed. Recent studies have indicated that bruxism is modulated by the central nervous system, though the neural mechanisms involved remain rarely studied (6, 7). The rationale behind this study was to use functional magnetic resonance imaging to design an experiment to observe possible functional changes in the cerebral cortex resulting from splint therapy based on the current knowledge on bruxism.

In clinical practice, occlusal splint therapy is the most common therapeutic approach for patients diagnosed with bruxism. Various types of splints are applied with varying durations. Occlusal splints are more likely to be used for the management of pain in the masticatory muscles, modification of the intermaxillary relationship, and adjustment of occlusal force distribution rather than to cure bruxism itself (7). The actual mechanism of efficacy remains debatable. Authors recommend that splint therapy with the behavioral approach as the most suitable modality for treatment (8).

The masticatory system is one of the oldest phylogenetic functional units and is a complex biological entity involving a highly coordinated central nervous system, peripheral effector organs, and sensory input (9). Rhythmic movements are established by 3 main structures: the motor cortex, which initiates and stops mastication, and also delivers pre-programmed movement patterns depending on the expectations and feedback; the central pattern generator, which provides basic rhythmic activity to jaw muscles; and the peripheral input, which may be the most powerful and variable input to the motoneurons (10). Research on mastication has quickly progressed in recent years with the aid of modern non-invasive neuro-imaging techniques. In a recent study, we performed a literature search within the PubMed and Web of Science databases with MeSH terms. A total of 2815 articles were found related to bruxism and 637 of these articles were related to sleep bruxism; 359 papers were published in the last 5 years and 27 of them were clinical research articles while 40 of them were reviews. Only 11 of the clinical research papers were controlled trials. This demonstrates that interest in bruxism has increased in recent years but controlled and evidence-based studies are few in number. A better understanding of the neurological basis for the masticatory parafunction is critical to develop an appropriate treatment. This includes identifying risk groups and protecting orofacial structures from long term damage. There are several studies on bruxism and brain activity in literature. The hypothesis of this experiment was that the use of occlusal splint in bruxism patients may change the activation in the functional areas which are different from healthy subjects.

MATERIALS and METHODS

Subjects

This study investigated a total of 30 volunteers between 20-27 years of age (mean age 23±1.85 years). All participants were right-handed females and had no history of neurological disorders, abnormalities of systemic function or contraindication for MRI, and had normal sleep, diet and healthy dentition with class I occlusion. The research diagnostic criteria for temporomandibular disorder (RDC/TMD) was used to exclude patients who had had myogenic and articular problems derived from TMD (11). 15 healthy participants were defined as a control group and 15 bruxism patients for a bruxism group based on the criteria which Lobbezoo et al. defined with an international consensus in 2013 (Table I) (6). Extraoral and intraoral examinations were performed for each participant by en expert clinician. The masseter hypertrophy, presence of tooth wear and fracture, and temporary masticatory muscle pain while awakening criteria were assessed. Bruxism splints were

prepared directly with the patient on the dental chair in accordance with the manufacturers 'instructions (Figure 1A-E). Written informed consent was obtained from each subject after the aims and the methodology were explained. This protocol was approved by the ethics committee of the medical faculty of Kırıkkale University, Turkey (decision number: 02/04), based on the guidelines set forth in the Declaration of Helsinki.

Experimental Protocol

Each participant received premagnet training before image acquisition. The task paradigm involved 5 motor tasks of isometric rhythmic voluntary tooth clenching while the mandible was at the maximal intercuspal position. Controlled measurement of muscle contraction was not carried out; patients were only trained about the clenching rhythm and position before imaging. Subjects were laid in the supine position on the scanner table with their heads immobilised with vacuum pads. The head coil and prismatic mirror were placed so that the task movie could be seen on the computer screen. All participants wore headphones for communication and disposable earplugs to minimise the noise heard in sequences. They were instructed to follow the task movie and perform the clenching task when the green spot appeared on-screen and rest when the red spot was seen. Furthermore, patients were informed not to make any motor movements including orofacial movements. The task paradigm was a block design alternation between 20 seconds of rest and 12 seconds of clenching (Figure 2). Each run consisted of 6 rest and 5 clench blocks. Scanning was performed 2 times for participants in the bruxism group (pre-treatment and post-treatment) and 1 time for the control group.

Image Acquisition

Magnetic resonance imaging was performed on a 3.0 Tesla scanner (Siemens Medical, Germany) with a 32 channel head coil. For each subject, a T_1 weighted 3D MPRAGE

Table I: The novel diagnostic grading system suggested by an international group of bruxism experts by a consensus at 2013 [Lobbezoo et al. 2013].

Diagnostic grading system for bruxism	
Possible bruxism	Sleep or awake bruxism should be based on self-report, by means of questionnaires and/or the anamnestic part of a clinical examination_
Probable bruxism	Self-reported sleep or awake bruxism supported with clinical findings of inspection
Definite bruxism	Self-reported sleep or awake bruxism, clinical findings and polysomnographic recordings

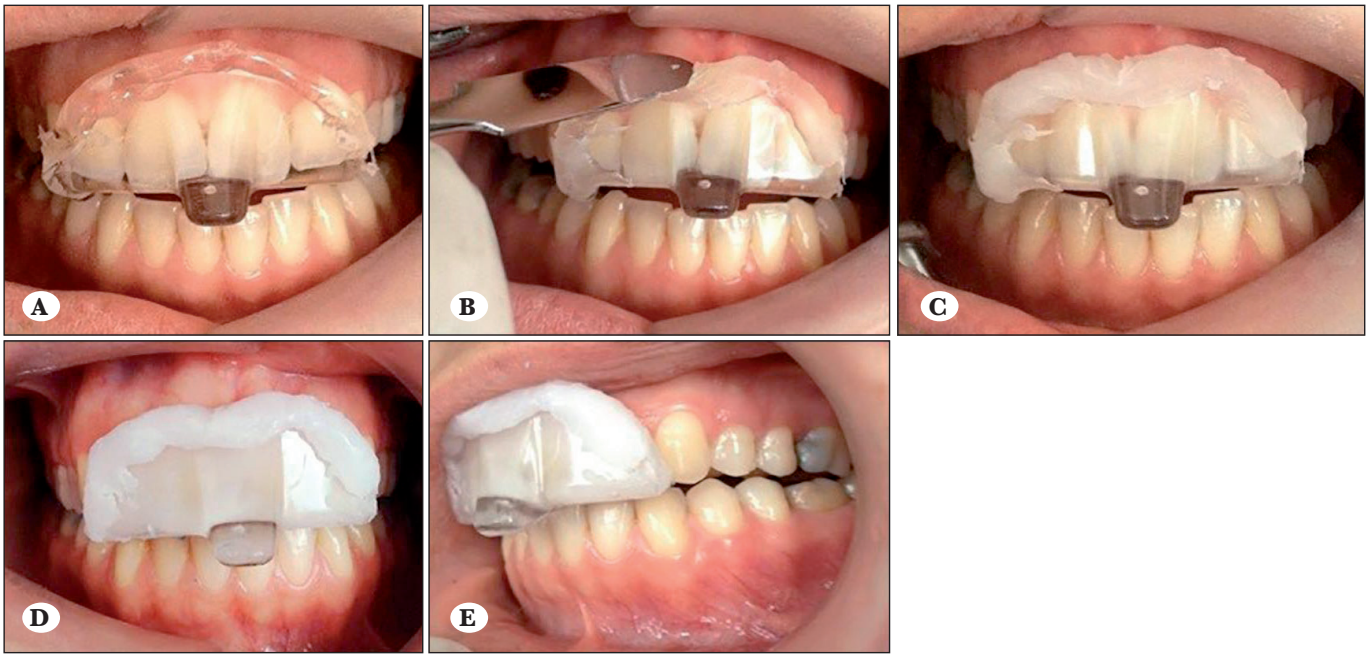

Figure 1: Chair-side preparation steps of NTI-tss splint by the clinician: **A)** Positioning the matrix on upper incisors loaded with thermoplastic filler which took a gel form in 70°C water. **B)** Adjusting the edges with lip movements and spatula **C)** Leaving the splint on incisors for about 5 minutes and then chilling in cold water **D)** Fitting and trimming. **E)** Confirming minimal space between the posterior teeth in lateral and protrusive movements.

sequence (TR = 2000 ms, TE = 35, slice thickness = 0,84 mm. Flip angle = 12, FOV = 215, matrix size = 256 x 256 x 256, 208 slices) high resolution anatomical scan was acquired before the functional imaging. The functional images consisted of echoplanar image volumes which were sensitive to BOLD (blood oxygenation level dependent) contrast (TR = 2000 ms, TE = 35, slice thickness = 3.0 mm. Flip angle = 75, FOV = 192, matrix size = 64 x 64. Number of volumes = 104, 28 slices).

Data Analysis

The functional magnetic resonance imaging (fMRI) data were evaluated using BrainVoyager QX 2.8 (Brain Innovation, Maastricht, Netherlands). During data analysis, the first 4 volumes were rejected because of signal instability. To eliminate motion artifacts, all data from subjects whose heads were observed to have moved more than 1.5 mm were discarded. Data series were motion corrected, spatial smoothing was performed, and images transformed to Talairach space by co-registration with Talairach-processed anatomical data. Activation maps were created using the General Linear Model: single study and the General Linear Model: multi study multi subject for statistical group analysis.

RESULTS

A statistical map of group analysis of the pre-treatment group minus the post-treatment group showed significantly less activation in the posterior cingulate cortex ($p<0.005$) (Figure 3, Table II). The areas represented Brodmann 31 and 23. In the second step analysis, the control group and the post-treatment group were compared and found to have no statistically significant difference which indicates increased BOLD signal in bruxism patients.

DISCUSSION

Previous research on bruxism and splint therapy has focused mainly on muscle activation changes that are measured by electromyography (12-15). Our study is based on the hypothesis that parafunctional activation of

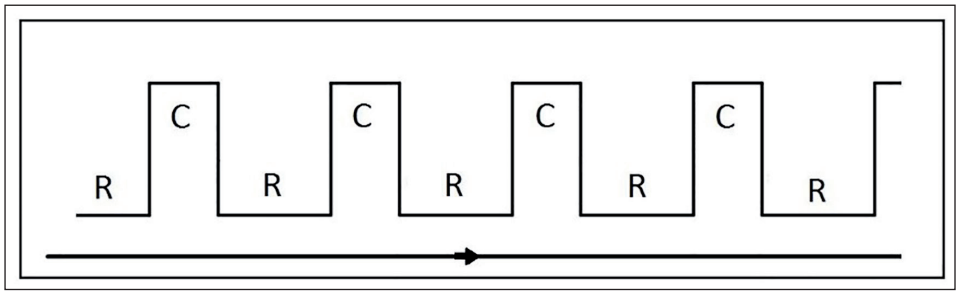

Figure 2: Task paradigm. R: rest; C: clench. 20 seconds of rest, 12 seconds of clenching periods.

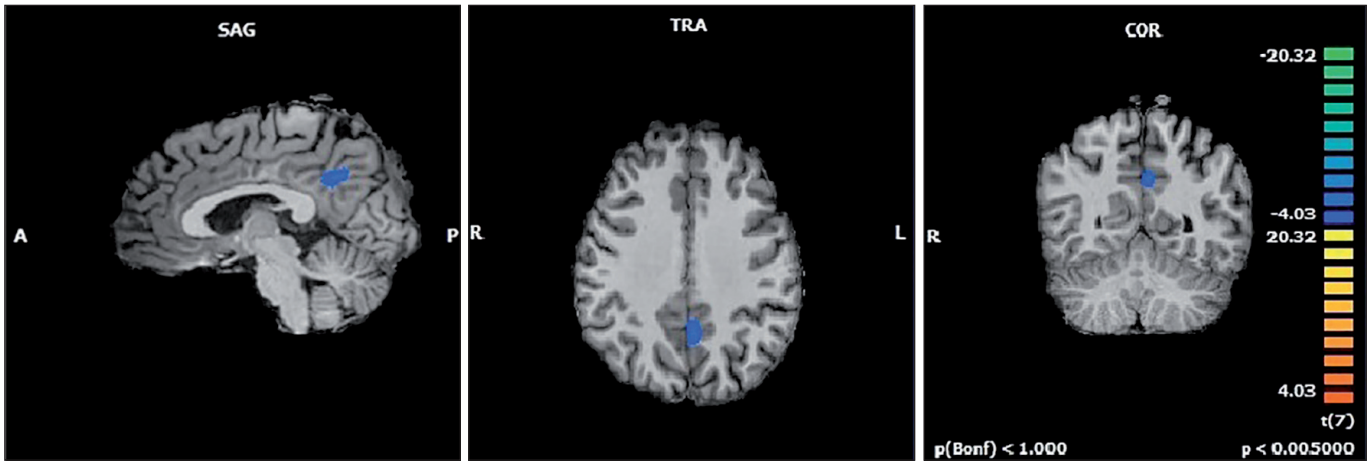

Figure 3: Post-treatment group minus pre-treatment group contrast. Blue cluster indicates location and size of less activated areas.

Table II: Talairach coordinates and activation peak values of pre- and post- treatment group contrast (BA= Brodmann Area). The values indicates increased BOLD signal activation size and location.

Anatomic landmarks	Side	peak x	peak y	peak z	t	p	Cluster size
Posterior cingulate cortex (BA 23,31)	Left	-4	-56	33	-7.64	0.000123	482

masticatory muscles is generated from the central nervous system and is supported by the fact that we observed cortical changes between healthy individuals and bruxism patients in clenching function in our preliminary experiment (16). Finding reduced mean percent signal changes in related cortical areas of bruxism patients relative to the same areas of control subjects supported this hypothesis. Proceeding from these findings, we designed this research to evaluate possible changes in cerebral cortex activation pattern in bruxism patients after occlusal splint therapy.

With the progress in technology besides high resolution anatomical imaging, it is possible to visualise systems and functions easily as well. It is important to understand masticatory function and parafunction which has crucial effects on the health of the entire body as well as its cognitive functions (17). Namely, the bruxism patients whose normal structure exposes a functional anomaly. The techniques in neuroscience used for functional imaging are transcranial magnetic stimulation, near infrared spectroscopy, PET, SPECT, EEG, MEG and fMRI. In our study. we used fMRI which is a widely used method for brain activation studies and has high temporal and spatial resolution.

In first step data analysis for each participant, we observed an activation pattern in the sensorimotor cortex and primary motor area similar to animal and human studies of the tooth clenching block (18-21).

One of the first neuroimaging studies which investigated group differences between healthy subjects and self-reported bruxism patients was performed by Wong et al. (22). They concluded that the control group revealed decreased activation areas in the supplementary motor area and inferior parietal lobule. These areas are associated with motor control of the biting force and mediate somatosensory feedback with which tactile proprioception is transferred (23, 24). Yılmaz found a similar decreased activation pattern in bruxism patients compared to the control group (16). Additionally, it is reported that these areas play a role in motor attention (25). The decreased activation in these areas compared to the control group might be due to diminished proprioceptive awareness (22).

It is believed that the somatosensory association area is in charge of visiomotor orientation. Brodmann 5 and 7, which is situated in this area, has a role in imitating motor learning circuits and motivation. Byrd et al. compared self-reporting bruxism patients and a control group and found hypoactivation in the motor cortex (supplementary motor area, sensorimotor area and rolandic operculum) and subcortical (caudate) areas in bruxist patients (26). The common function of these areas might be movement guidance. In our study, we performed functional imaging before and after splint therapy and observed incremental activation in the inferior parietal lobule and dorsal posterior cingulate area. This increase in activation pattern should be derived from motor learning and habituation. Behr et al. mentioned that neuroplasticity could be related to bruxism (27). It has been demonstrated that after a period of time with continued exercises, masticatory muscles are significantly prone to motor adaptation in the central nervous system (28, 29). These findings may offer the possibility that splint therapy causes an increase of activation in the areas that guide masticatory movements.

Lickteig et al. performed an fMRI investigation with a Michigan type splint used for 11 days and suggested that splint therapy triggered activation in the parietal sensorimotor integration areas (30). In our study, we informed the patients about to use the NTI-tss splint for 11 nights at least 8 hours a day about the risk of occlusal changes in long term use. As far as we are aware, the adequate use of the NTI splint for bruxism is not described in the literature.

Splint therapy data analysis between pre- and post-treatment groups revealed similar results to those identified in previous studies as a significant signal increase in activation pattern in the posterior cingulate cortex (Brodmann 23) (22, 26).

Researchers who have worked on non-human primates have suggested that the excitability of corticobulbar pathways is depressed during sleep and the rhythmic masticatory activity during sleep may not be under the direct influence of the cerebral cortex (31-33). In this study, we observed both increased and decreased areas of activation in subcortical areas individually. The potential involvement of the brainstem with bruxism has not been elucidated because of the technical problems with imaging artefacts originating from cardiac and respiratory functions and head motions during sleep (26).

Studies investigating the mastication task with fMRI noted that changes with age could affect BOLD signal intensity, so we limited the age interval to between 20 and 27 (19). Furthermore, to maintain an uncomplicated data analysis due to brain volumes, we preferred to work only with right handed female volunteers.

Functional MRI requires considerable thoroughness in technical terms. Subjects should not perform any motor function including those in the orofacial region except for the imaging task. In our study, 2 patients in the bruxism group developed phobia in the gantry and 1 patient was excluded due to motion artefact. In the control group, 1 subject left the study because of medical problems. In order to equalize the number of individuals from both groups, data analysis was continued over 12 persons for each group.

We detected extracranial activation patterns in some functional images. We are in agreement with previous reports that temporalis muscle activity could be detected on the fMRI scans (34).

Limitations of this study were as follows: we made a diagnosis of bruxism by clinical examination and patient history according to the criteria stated by Lobbezoo et. al. (6). The second limitation is that we could not find an evidence-based duration of NTI splint use for bruxism in the literature. We therefore designed the experiment based on a similar study (30). Finally, the analysis of functional data requires proficiency. We performed some analyses repeatedly and excluded some data to work on uncorrupted results.

Based on the present objective fMRI findings, we suggest that there are significant differences between healthy subjects and participants with bruxism. Eleven days of splint therapy may be recommendable for the management of bruxism, parallel with the prevailing opinion that supports splint therapy with behavioral therapy. However the mechanisms underlying this parafunction require further research.

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