

RESEARCH ARTICLE

To see bruxism: a functional MRI study

S Yılmaz

Department of Oral and Maxillofacial Radiology, Faculty of Dentistry, Kirikkale University, Kirikkale, Turkey

Objective: Since the pathophysiology of bruxism is not clearly understood, there exists no possible treatment. The aim of this study is to investigate the cerebral activation differences between healthy subjects and patients with bruxism on behalf of possible aetiological factors.

Methods: 12 healthy subjects and 12 patients with bruxism, a total of 24 right-handed female subjects (aged 20–27 years) were examined using functional MRI during tooth-clenching and resting tasks. Imaging was performed with 3.0-T MRI scanner with a 32-channel head coil. Differences in regional brain activity between patients with bruxism and healthy subjects (control group) were observed with BrainVoyager QX 2.8 (Brain Innovation, Maastricht, Netherlands) statistical data analysis program. Activation maps were created using the general linear model: single study and multistudy multisubject for statistical group analysis. This protocol was approved by the ethics committee of medical faculty of Kirikkale University, Turkey (02/04), based on the guidelines set forth in the Declaration of Helsinki.

Results: The group analysis revealed a statistically significant increase in blood oxygenation level-dependent signal of three clusters in the control group ($p < 0.005$), which may indicate brain regions related with somatognosis, repetitive passive motion, proprioception and tactile perception. These areas coincide with Brodmann areas 7, 31, 39 and 40. It is conceivable that there are differences between healthy subjects and patients with bruxism.

Conclusions: Our findings indicate that there was a decrease of cortical activation pattern in patients with bruxism in clenching tasks. This indicates decreased blood flow and activation in regional neuronal activity. Bruxism, as an oral motor disorder concerns dentistry, neurology and psychiatry. These results might improve the understanding and physiological handling of sleep bruxism.

Dentomaxillofacial Radiology (2015) **44**, 20150019. doi: [10.1259/dmfr.20150019](https://doi.org/10.1259/dmfr.20150019)

Cite this article as: Yılmaz S. To see bruxism: a functional MRI study. *Dentomaxillofac Radiol* 2015; **44**: 20150019.

Keywords: bruxism; fMRI; masticatory system; brain mapping

Introduction

Bruxism is a parafunctional masticatory activity associated with tooth clenching and grinding, which affects millions of people worldwide.¹ This oral motor disorder concerns many disciplines, including dentistry, neurology and psychiatry.^{2,3} Occlusal splints are commonly used as a treatment choice for patients with bruxism in clinical practice. Because the pathophysiology of the disorder is not yet clearly understood, there is no accurate treatment to date. Many practitioners believe that bruxism is caused by psychological stress, but such statements are

not strongly evidence based.^{4,5} The majority of patients with sleep bruxism represent any associated medical or psychiatric conditions (idiopathic or primary bruxism), while patients with daytime bruxism have been reported to depict basal ganglia infarction, multisystem atrophy, cervical dystonia, or use and deprivation of some drugs (secondary bruxism).^{6,7} There are various explanations on the aetiology and pathophysiology of this disorder in literature, but none of them is confirmed or disproved scientifically, yet. Currently, the focus of aetiology is more on the mechanisms of the central nervous system. In addition, bruxism appears to be modulated by neurophysiological and behavioural factors.⁸ The

Correspondence to: Dr Selmi Yılmaz. E-mail: selmiyard@gmail.com

Received 18 January 2015; revised 20 March 2015; accepted 24 March 2015

influence of cerebral circuits on occlusal movements is unknown, and it has been suggested that possible benefits of an occlusal splint arise from the presence of a foreign object, consciousness or cerebral learning.^{9,10}

MRI is a sophisticated technique that presents structural anatomy of the brain with high spatial resolution. Functional MRI (fMRI) provides anatomical data in addition with functional data. By using MR principles, mapping of brain activation in a specific time period can be performed.¹¹ Recently, several investigations about detecting activation during chewing in healthy subjects showed cortical activation mainly in bilaterally somatosensory (S1) and motor cortex (M1), secondary somatosensory (S2), premotor cortex, supplementary motor area, parietal cortex, insula, thalamus and cerebellum, which demonstrates a role for masticatory activity in maintaining the homeostasis of the body and brain function.^{10,12,13} There are several studies about bruxism and brain activity in literature.^{12,14,15} Based on the recent idea of the influence of central mechanisms on bruxism, we aimed to investigate the functional differences between patients with bruxism diagnosed with the renovated diagnostic criteria and healthy subjects with 3.0-T fMRI.

Methods and materials

Subjects

In this study, 30 volunteers were included in the experiment, aged between 20 and 27 years (mean age, 23 ± 1.85 years). After experimental protocol and data analysis, 24 female subjects finally participated in the study. All participants were right-handed females. They had normal sleeping and eating habits and had no neurological disorders, abnormalities of systemic function or contraindication for MRI in their medical histories. All participants were asked if they had any operation in the past 6 months that left their mouth open for a long time. Extraoral and intraoral examinations were performed for each participant. Research diagnostic criteria for temporomandibular disorder (TMD) were used to exclude patients with myogenic and articular problems derived from TMD.¹⁶ Bruxism was identified based on the criteria that Lobbezoo et al¹⁷ defined with an international consensus in 2013. Also, all participants had healthy dentition with Class 1 occlusion. Using these criteria, 15 healthy subjects were defined as the control group and 15 patients with “probable” bruxism as the bruxism group. 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 medical faculty of Kirikkale University, Turkey (project number 02/04), based on the guidelines set forth in the Declaration of Helsinki.

Experimental protocol

Each participant had pre-magnet training before fMRI. They were instructed to follow the specially prepared task movie and perform the clenching task when a green

spot appears on the screen and rest when a red spot is seen. The explained task paradigm involved five motor tasks of isometric rhythmic voluntary tooth clenching when the mandible was at the maximal intercuspal position and six rest tasks with the mandible positioned as to maintain freeway space in the resting position. Task paradigm was a block design that alternates between 20 s of rest and 12 s of clenching period (Figure 1). Subjects were laid in the supine position on the scanner table, and their head immobilized with vacuum pads. Head coil and prismatic mirror were placed so the computer screen projection could be seen easily. All participants wore disposable earplugs to minimize the noise heard in image acquisition sequences and headphones for communication. Furthermore, patients were informed not to make any motor movements including the orofacial area.

Image acquisition

MRI was performed on a 3.0-T scanner (Siemens Medical, Erlangen, Germany) with a 32-channel head coil. Before functional imaging, a T_1 weighted three-dimensional MPRAGE (magnetization prepared rapid acquisition gradient recalled echo) sequence (repetition time, 2000 ms; echo time, 35 ms; matrix size, $256 \times 256 \times 256$; slice thickness, 0.84 mm; flip angle, 12° ; field of view, 215; 208 slices) high-resolution anatomical scan was acquired for each subject. The functional images consisted of echoplanar image volumes that were sensitive to blood oxygenation level-dependent (BOLD) contrast (repetition time, 2000 ms; echo time, 35 ms; matrix size, 64×64 ; slice thickness, 3.0 mm; flip angle, 75° ; field of view, 192; number of volumes, 104; 28 slices).

Data analysis

The fMRI data for each participant were evaluated using BrainVoyager QX 2.8 (Brain Innovation, Maastricht, Netherlands) program. Owing to signal instability, the first four volumes were omitted from the data analysis. To eliminate motion artefacts, subjects whose heads displaced >1.5 mm were discarded. Data series were motion corrected, and spatial smoothing was performed and transformed to Talairach space by

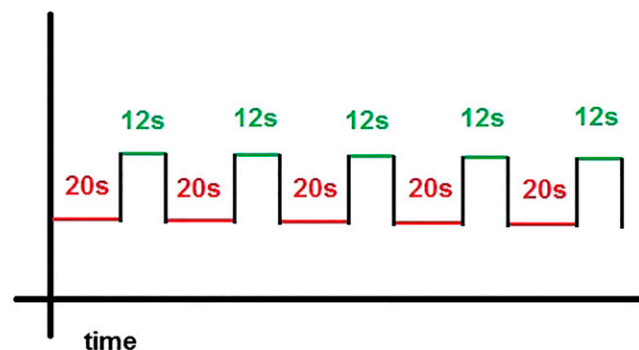


Figure 1 Task paradigm. One session consists of 20 s of rest and continues with 12 s of clenching period.

co-registration with Talairach-processed anatomical data. Activation maps were created using the general linear model—single study—and the general linear model—multistudy multisubject—for statistical group analysis.

Results

Motion correction, temporal high-pass filtering and intensity inhomogeneity correction steps were applied to each data to minimize artefacts. For each participant individually, activation maps of clenching minus rest BOLD signal were calculated (Figure 2). Statistical maps of group analysis showed significant increase in BOLD signal in the control group compared with the bruxism group ($p < 0.005$) (Figure 3). The bruxism group showed less activation in the right inferior parietal lobule [Brodmann areas (BA) 39, 40 and partially 7] and dorsal posterior cingulate area (BA 31). The locations of the most significant foci of activation for group analysis are summarized in Table 1 with landmarks and Talairach co-ordinates.

Discussion

Bruxism is still an unsolved problem in dentistry. Rehabilitation of this dysfunction can contribute to the enhancement of life expectancy, whole-body health and life quality. In diagnostic approach, it must be noted that pathologies with symptoms in the orofacial region can originate from different mechanisms of different systems. In this study, the hypothesis is that parafunctional activation of masticatory muscles is generated from the central nervous system; therefore, we aimed to observe cortical changes between healthy controls and patients with bruxism in mastication function.

In dentomaxillofacial radiology, the techniques commonly used are plain radiography, CT, CBCT, MRI and ultrasound. With the progress in technology together with high-resolution anatomical imaging, it is possible to also visualize systems and functions easily. In literature, there are only a few studies in the area of human research that focus on interactions between occlusion and brain function. It is important to understand masticatory function, and also parafunction that has crucial effects on whole-body health as well as

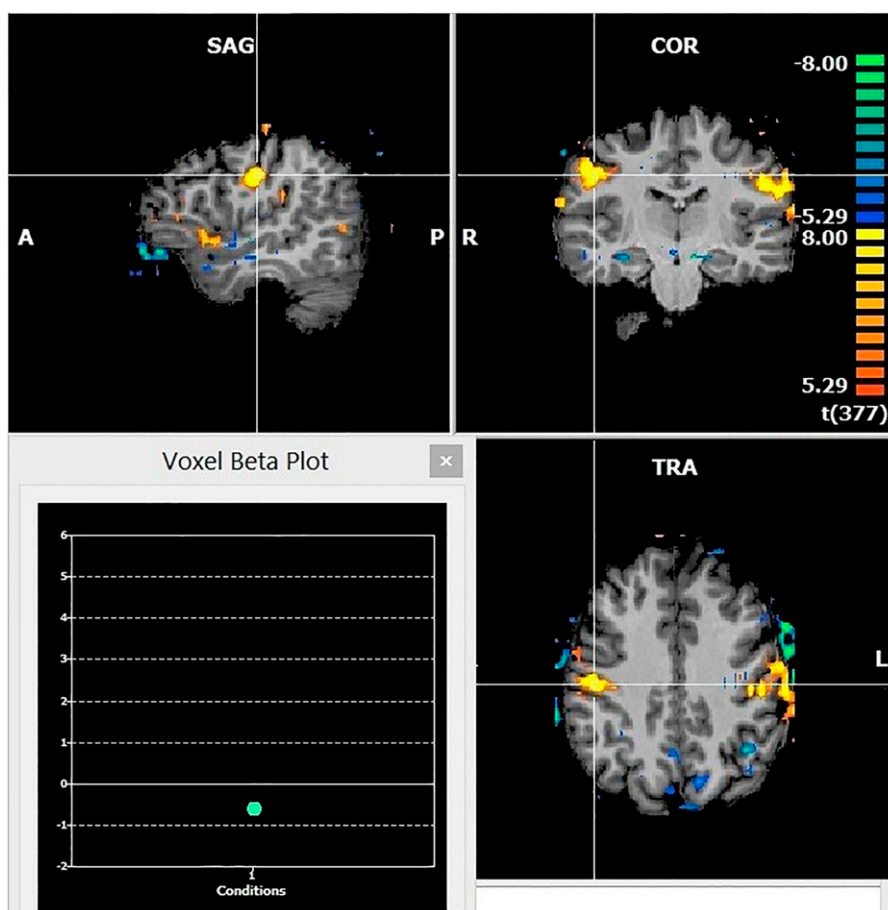


Figure 2 Blood oxygenation level-dependent signal differences between rest and clenching periods. Yellow areas show increased activations during tooth clenching block. These areas are in accordance with cortical mastication area. A, anterior; COR, coronal; L, left; P, posterior; R, right; SAG, sagittal; TRA, transversal. For colour images please see online.

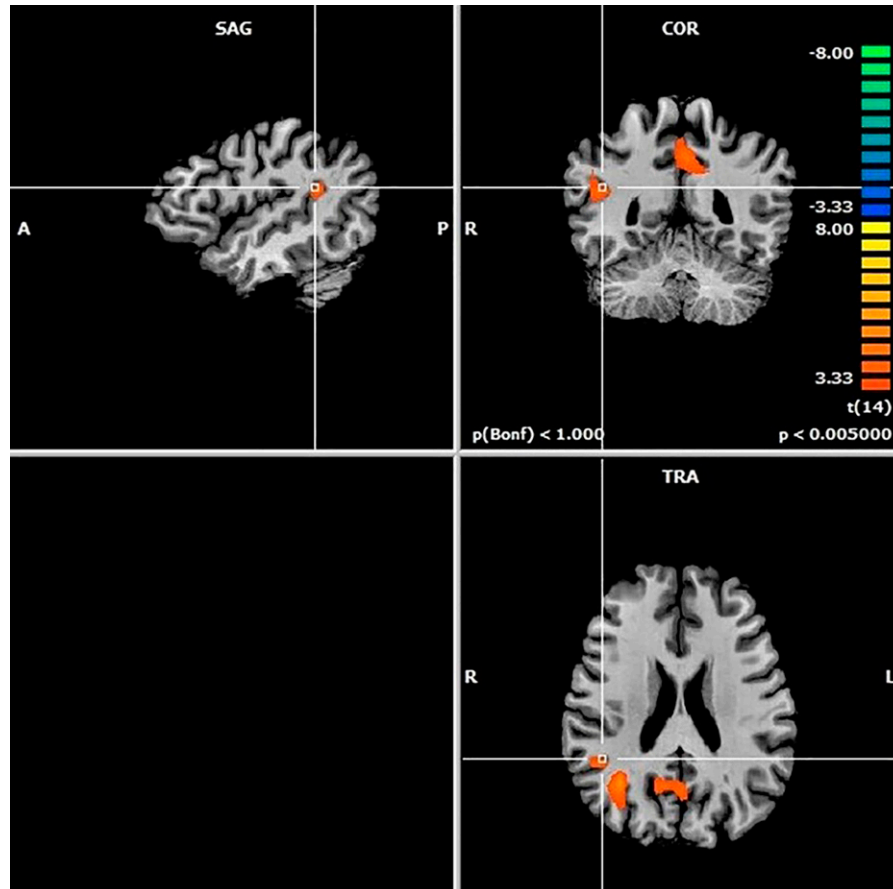


Figure 3 Cross-group activation differences. Control group minus bruxism group. Three clusters of increases in blood oxygenation level-dependent signal in the control group is observed. A, anterior; COR, coronal; L, left; P, posterior; R, right; SAG, sagittal; TRA, transversal.

cognitive functions.¹⁸ By the end of the 20th century, non-invasive measurements of brain function were developed.¹⁹ The fMRI technique was first used in a study published in 1991, which showed how a standard MRI scanner can be used to track where oxygenated and deoxygenated blood flows in the brain.²⁰ In first-step data analysis for each participant, we calculated BOLD signal changes between clenching and rest blocks and observed activation pattern in the cortical mastication area as described in previous studies (Figures 2 and 4).^{21–24}

Byrd et al¹⁴ compared self-reporting patients with bruxism and the control group using fMRI. They found hypoactivation in the motor cortex (supplementary motor area, sensorymotor area and rolandic operculum) and the subcortical (caudate) areas in patients with bruxism in parafunctional movements. The common function of these areas might be movement guidance. After cross-group comparisons, our results revealed increased activation pattern in the control group in the inferior parietal lobule, which was implicated as a possible role of sensorimotor integration during orofacial movements (Figure 3). Wong et al¹² concluded that the control group revealed activation areas in the supplementary motor area and inferior parietal lobule

in patients with bruxism and the control group, similar to our study. It is reported that these areas play a role in motor attention, motor control of biting force and mediating somatosensory feedback where tactile proprioception is transferred.^{25–27} The decreased activation in these areas compared with those of the control group might be owing to diminished proprioceptive awareness in patients with bruxism.¹²

In previous studies that worked with bruxism, patient groups were selected by self-reports of tooth grinding/clenching or questionnaires, which provide inadequate information for diagnosis of bruxism.^{12,14} In our study, we selected the bruxism group according to novel diagnostic criteria that were defined by the consensus that was chaired by Lobbezoo et al¹⁷ in 2013.

In a previous report on the brain activity associated with clenching and chewing task, Kervancioglu et al¹⁵ applied magnetoencephalography and found that patients with bruxism revealed increased cortical activation in BA 4, the primary somatomotor area, when compared with healthy controls. The results of a recent study do not completely agree with these reports; for example, when we applied cross-group analysis as control group minus bruxism group, increased activation in

Table 1 Talairach co-ordinates and activation peak values of significantly activated regions in the group analysis between the bruxism group and control group; clenching minus baseline

Anatomic landmark	Side	Peak x	Peak y	Peak z	t	p-value	Cluster size (total voxels)
Dorsal posterior cingulate area (BA 31)	Right	35	-59	24	5.67	0.000058	632
Inferior parietal lobule (BA 39)	Right	-1	-65	24	4.56	0.000443	476
Inferior parietal lobule (BA 40)	Right	-4	-47	42	4.66	0.000366	390

BA, Brodmann area.

the control group in three clusters was found. They explained that increased activity was owing to increased sensorimotor cortical representation of the tongue and chewing muscles depending on enhanced parafunctional muscle activity in patients with bruxism triggered by occlusal factors.²⁷ However, recent literature contains insufficient evidence to support the role of occlusion in the genesis of bruxism.²⁸

In a recent study, subjects executed voluntary isometric muscle contraction with the clenching task. The controlled measurement of masticatory contraction was not carried out. In previous fMRI reports, in which isotonic muscle contraction was performed with gum-chewing and tooth-tapping tasks, much wider areas of activation were demonstrated in the cortex than in isometric contraction tasks.^{13,21,22,24,29} Additionally, no major differences were observed in brain activity within low levels of tooth clenching with controlled force.³⁰

Quintero et al²⁴ investigated brain activity during gum-chewing task and found activation in the cerebellum, which is involved in the co-ordination and rhythmicity of oral functions as well as activity in the cortical and subcortical areas. However, in a previous magnetoencephalography study, researchers could not find significant activation patterns in the cerebellum between the bruxism and control groups during the tooth-clenching task.¹⁵ We did not focus on the cerebellum as a region of interest, but in initial analysis for individuals, some images represented decreased and increased BOLD signal areas in the cerebellum and subcortical areas. Brainstem regions are involved as central pattern generators and timing networks in mastication.³¹

Several limitations of this study should be mentioned. Firstly, fMRI is a method that requires a high degree of attention and care in experimental paradigm, patient attunement and statistical data analysis. We started our experiment with 15 patients in each group but

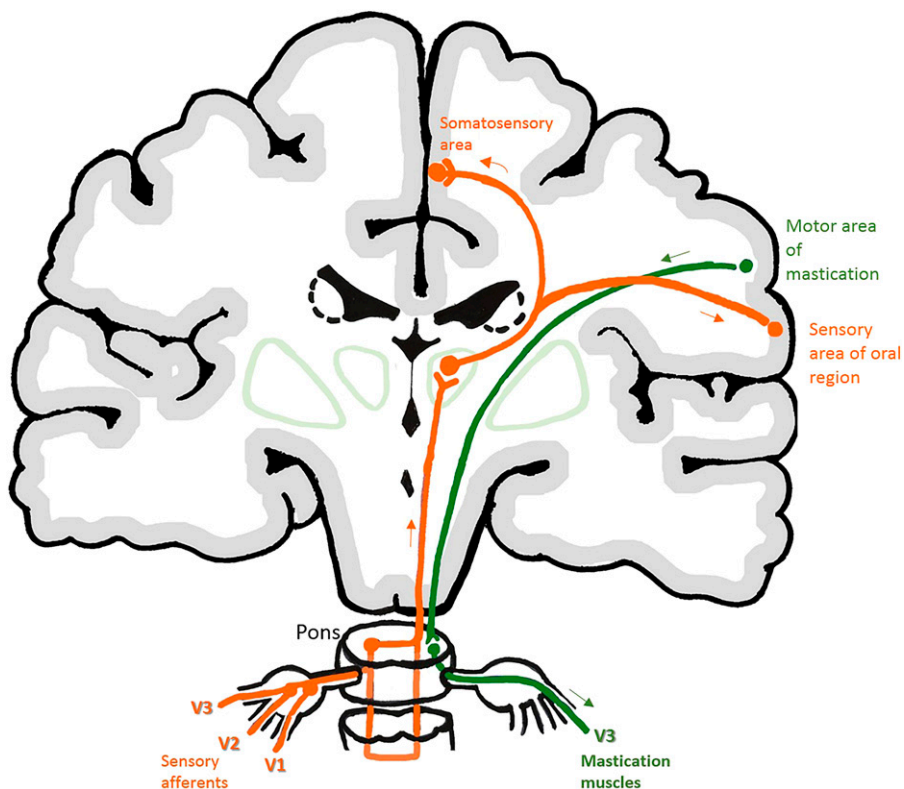


Figure 4 Motor and sensory component of trigeminal nerve and cortical mastication areas. V1, first branch of fifth cranial nerve; V2, second branch of fifth cranial nerve; V3, third branch of fifth cranial nerve.

conducted with 12 patients for each group; as a consequence of 2 patients having anxiety in gantry and 1 patient showing motion artefact in data analysis in the bruxism group. Also, a patient had to leave the study because of health problems. Therefore, we equalized the group numbers for statistical analysis. The number of the participants was enough when we compared with similar studies.^{12,14,15} Also, we limited the age group between 20 and 27 years because of the changes that occur in the brain with ageing. It has been mentioned that ageing generates a decrease in prevalence in bruxism. We preferred to study the stated age group because in clinical practice, bruxism is frequently observed between the ages of 18 and 29 years.^{32,33} Likewise, increased age causes a decrease in BOLD signal.³⁴ Additionally, we chose all participants to be female because of the possible statistical difficulties that might be derived from brain volume differences between the genders.

Another functional imaging modality, diffusion tensor imaging, which enables functional connectivity of

the white matter between different functional areas, might be useful to see whether there are differences in the structural connectivity between patients with bruxism and healthy controls and, if so, whether these differences are reduced after the therapy.

Further studies should investigate patients with bruxism diagnosed with polysomnographic records supported with video and voice records in larger population and both gender groups. As a result of our study and existing knowledge, the use of fMRI is clinically important, and it is a new opportunity in diagnosis, evaluation, management and follow-up of the treatment of bruxism as well as orofacial pain conditions for clinicians in dentistry, neurology and psychiatry. Precise identification of brain activation pattern related to bruxism during sleep can be obtained when a neuro-imaging technique is developed that tolerates head movements during sleep.

References

1. Strausz T, Ahlberg J, Lobbezoo F, Restrepo CC, Hublin C, Ahlberg K, et al. Awareness of tooth grinding and clenching from adolescence to young adulthood: a nine-year follow-up. *J Oral Rehabil* 2010; **37**: 497–500. doi: [10.1111/j.1365-2842.2010.02071.x](https://doi.org/10.1111/j.1365-2842.2010.02071.x)
2. Giraki M, Schneider C, Schäfer R, Singh P, Franz M, Raab WH, et al. Correlation between stress, stress-coping and current sleep bruxism. *Head Face Med* 2010; **6**: 2. doi: [10.1186/1746-160X-6-2](https://doi.org/10.1186/1746-160X-6-2)
3. Ommerborn MA, Schneider C, Giraki M, Schäfer R, Handschel J, Franz M, et al. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. *Eur J Oral Sci* 2007; **115**: 7–14. doi: [10.1111/j.1600-0722.2007.00417.x](https://doi.org/10.1111/j.1600-0722.2007.00417.x)
4. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. *Chest* 2001; **119**: 53–61. doi: [10.1378/chest.119.1.53](https://doi.org/10.1378/chest.119.1.53)
5. Manfredini D, Landi N, Romagnoli M, Bosco M. Psychic and occlusal factors in bruxers. *Aust Dent J* 2004; **49**: 84–9. doi: [10.1111/j.1834-7819.2004.tb00055.x](https://doi.org/10.1111/j.1834-7819.2004.tb00055.x)
6. Raigrodski AJ, Mohamed SE, Gardiner DM. The effect of amitriptyline on pain intensity and perception of stress in bruxers. *J Prosthodont* 2001; **10**: 73–7. doi: [10.1111/j.1532-849X.2001.00073.x](https://doi.org/10.1111/j.1532-849X.2001.00073.x)
7. Jaffee MS, Bostwick JM. Buspirone as an antidote to venlafaxine-induced bruxism. *Psychosomatics* 2000; **41**: 535–6. doi: [10.1176/appi.psy.41.6.535](https://doi.org/10.1176/appi.psy.41.6.535)
8. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 2001; **28**: 1085–91. doi: [10.1046/j.1365-2842.2001.00839.x](https://doi.org/10.1046/j.1365-2842.2001.00839.x)
9. Baad-Hansen L, Jadidi F, Castrillon E, Thomsen PB, Svensson P. Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. *J Oral Rehabil* 2007; **34**: 105–11. doi: [10.1111/j.1365-2842.2006.01717.x](https://doi.org/10.1111/j.1365-2842.2006.01717.x)
10. Lickteig R, Lotze M, Lucas C, Domin M, Kordass B. Changes in cortical activation in craniomandibular disorders during splint therapy—a single subject fMRI study. *Ann Anat* 2012; **194**: 212–15. doi: [10.1016/j.aanat.2011.10.006](https://doi.org/10.1016/j.aanat.2011.10.006)
11. Huettel SA, Song AW, McCharty G. *Functional magnetic resonance imaging*. 2nd edn. Sunderland, MA: Sinauer Associates Inc.; 2009.
12. Wong D, Dziedzic M, Talavage TM, Romito LM, Byrd KE. Motor control of jaw movements: an fMRI study of parafunctional clench and grind behavior. *Brain Res* 2011; **1383**: 206–17. doi: [10.1016/j.brainres.2011.01.096](https://doi.org/10.1016/j.brainres.2011.01.096)
13. Iida T, Kato M, Komiyama O, Suzuki H, Asano T, Kuroki T, et al. Comparison of cerebral activity during teeth clenching and fist clenching: a functional magnetic resonance imaging study. *Eur J Oral Sci* 2010; **118**: 635–41. doi: [10.1111/j.1600-0722.2010.00784.x](https://doi.org/10.1111/j.1600-0722.2010.00784.x)
14. Byrd KE, Romito LM, Dziedzic M, Wong D, Talavage TM. fMRI study of brain activity elicited by oral parafunctional movements. *J Oral Rehabil* 2009; **36**: 346–61. doi: [10.1111/j.1365-2842.2009.01947.x](https://doi.org/10.1111/j.1365-2842.2009.01947.x)
15. Kervancioglu BB, Teismann IK, Rain M, Hugger S, Boeckmann JA, Young P, et al. Sensorimotor cortical activation in patients with sleep bruxism. *J Sleep Res* 2012; **21**: 507–14. doi: [10.1111/j.1365-2869.2012.01005.x](https://doi.org/10.1111/j.1365-2869.2012.01005.x)
16. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 1992; **6**: 301–55.
17. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil* 2013; **40**: 2–4. doi: [10.1111/joor.12011](https://doi.org/10.1111/joor.12011)
18. Ono Y, Yamamoto T, Kubo KY, Onozuka M. Occlusion and brain function: mastication as a prevention of cognitive dysfunction. *J Oral Rehabil* 2010; **37**: 624–40. doi: [10.1111/j.1365-2842.2010.02079.x](https://doi.org/10.1111/j.1365-2842.2010.02079.x)
19. Ohkubo C, Morokuma M, Yoneyama Y, Matsuda R, Lee JS. Interactions between occlusion and human brain function activities. *J Oral Rehabil* 2013; **40**: 119–29. doi: [10.1111/j.1365-2842.2012.02316.x](https://doi.org/10.1111/j.1365-2842.2012.02316.x)
20. Belliveau JW, Kennedy DN Jr, McKinstry RC, Buchbinder BR, Weisskoff RM, Cohen MS, et al. Functional mapping of the human visual cortex by magnetic resonance imaging. *Science* 1991; **5032**: 716–19. doi: [10.1126/science.1948051](https://doi.org/10.1126/science.1948051)
21. Onozuka M, Fujita M, Watanabe K, Hirano Y, Niwa M, Nishiyama K, et al. Mapping brain region activity during chewing: a functional magnetic resonance imaging study. *J Dent Res* 2002; **81**: 743–6. doi: [10.1177/154405910208101104](https://doi.org/10.1177/154405910208101104)
22. Tamura T, Kanayama T, Yoshida S, Kawasaki T. Functional magnetic resonance imaging of human jaw movements. *J Oral Rehabil* 2003; **30**: 614–22. doi: [10.1046/j.1365-2842.2003.01054.x](https://doi.org/10.1046/j.1365-2842.2003.01054.x)
23. Hiraba H, Sato T. Cortical control of mastication in cats. 2. Deficits of masticatory movements following a lesion in the motor cortex. *Somatosens Mot Res* 2005; **22**: 183–92. doi: [10.1080/08990220500262307](https://doi.org/10.1080/08990220500262307)
24. Quintero A, Ichescu E, Myers C, Schutt R, Gerstner GE. Brain activity and human unilateral chewing: an FMRI study. *J Dent Res* 2013; **92**: 136–42. doi: [10.1177/0022034512466265](https://doi.org/10.1177/0022034512466265)

25. Takada T, Miyamoto T. A fronto-parietal network for chewing of gum: a study on human subjects with functional magnetic resonance imaging. *Neurosci Lett* 2004; **360**: 137–40. doi: [10.1016/j.neulet.2004.02.052](https://doi.org/10.1016/j.neulet.2004.02.052)
26. Takahashi T, Miyamoto T, Terao A, Yokoyama A. Cerebral activation related to the control of mastication during changes in food hardness. *Neuroscience* 2007; **145**: 791–4. doi: [10.1016/j.neuroscience.2006.12.044](https://doi.org/10.1016/j.neuroscience.2006.12.044)
27. Rushworth MF, Nixon PD, Renowden S, Wade DT, Passingham RE. The left parietal cortex and motor attention. *Neuropsychologia* 1997; **35**: 1261–73. doi: [10.1016/s0028-3932\(97\)00050-x](https://doi.org/10.1016/s0028-3932(97)00050-x)
28. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. *J Oral Rehabil* 2008; **35**: 476–94. doi: [10.1111/j.1365-2842.2008.01881.x](https://doi.org/10.1111/j.1365-2842.2008.01881.x)
29. Tamura T, Kanayama T, Yoshida S, Kawasaki T. Analysis of brain activity during clenching by fMRI. *J Oral Rehabil* 2002; **29**: 467–72. doi: [10.1046/j.1365-2842.2002.00880.x](https://doi.org/10.1046/j.1365-2842.2002.00880.x)
30. Iida T, Overgaard A, Komiyama O, Weibull A, Baad-hansen L, Kawara M, et al. Analysis of brain and muscle activity during low-level tooth clenching—a feasibility study with a novel biting device. *J Oral Rehabil* 2014; **41**: 93–100. doi: [10.1111/joor.12128](https://doi.org/10.1111/joor.12128)
31. Türker KS. Reflex control of human jaw muscles. *Crit Rev Oral Biol Med* 2002; **13**: 85–104.
32. Bader G, Lavigne G. Sleep bruxism; an overview of an oro-mandibular sleep movement disorder. *Sleep Med Rev* 2000; **4**: 27–43. doi: [10.1053/smr.1999.0070](https://doi.org/10.1053/smr.1999.0070)
33. de la Hoz-Aizpurua JL, Diaz-Alonso E, LaTouche-Arbizu R, Mesa-Jiménez J. Sleep bruxism. Conceptual review and update. *Med Oral Patol Oral Cir Bucal* 2011; **16**: 231–8. doi: [10.4317/medoral.16.e231](https://doi.org/10.4317/medoral.16.e231)
34. Onozuka M, Fujita M, Watanabe K, Hirano Y, Niwa M, Nishiyama K, et al. Age-related changes in brain regional activity during chewing: a functional magnetic resonance imaging study. *J Dent Res* 2003; **82**: 657–60. doi: [10.1177/154405910308200817](https://doi.org/10.1177/154405910308200817)