Bruxism and the Effectiveness of Treatment with Mouth Guards versus Botulinum Toxin Type A

Amanda Lans Valera
02/07/13
3802957

Table of contents

Introduction

Bruxism is a condition illustrated with symptoms such as involuntary grinding, gnashing or the clenching of teeth with the use of the jaw muscle, or masseter. Most cases of bruxism occur during sleep, however, it should not be excluded as a daytime condition. Some forms of the condition are mild and thus do not require treatment, yet it can also occur that patients suffer from complaints such as headaches up to permanent jaw disorders known as oromandibular conditions (Mayo Clinic 2011). It might take months or years until complications develop. The disorder is often treated with a mouth guard, but recently botulinum toxin A is also used to temporarily and partially paralyzing the superficial and deep masseter muscles which are involved in bruxism. The paper aims to highlight current research on the topic and give an impression of the pros and cons of different treatment methods. The paper outlines the anatomical, physiological and psychosocial factors of bruxism as well as the consequences of bruxism.

Anatomy and Physiology

Bruxism can be seen as a disorder of repetitive, unconscious contraction of the masseter muscle. The masseter muscle can be divided into two sections, a superficial one and deep section. The superficial section, which is also the larger of the two, arises from a dense tendinous aponeurosis of the zygomatic process of the maxilla, and from the frontal two-thirds of the inferior edge of the zygomatic arch. The fibers of the muscle descend and move posteriorly in order to fit into the angle of lesser half of the lateral surface of the ramus of the mandible. The smaller section of the muscle, the deep section, is much more muscular and has more texture to it. It originates from the posterior third of the lower frame and from the whole of the medial surface of the zygomatic arch. Its muscular fibers descend and move anteriorly in order to fit into the upper half of the ramus and the lateral surface of the coronoid process of the mandible. The deep sections of the masseter muscle are partially hidden behind the superficial sections and the parotid gland (Grays 2010). See image A and B.

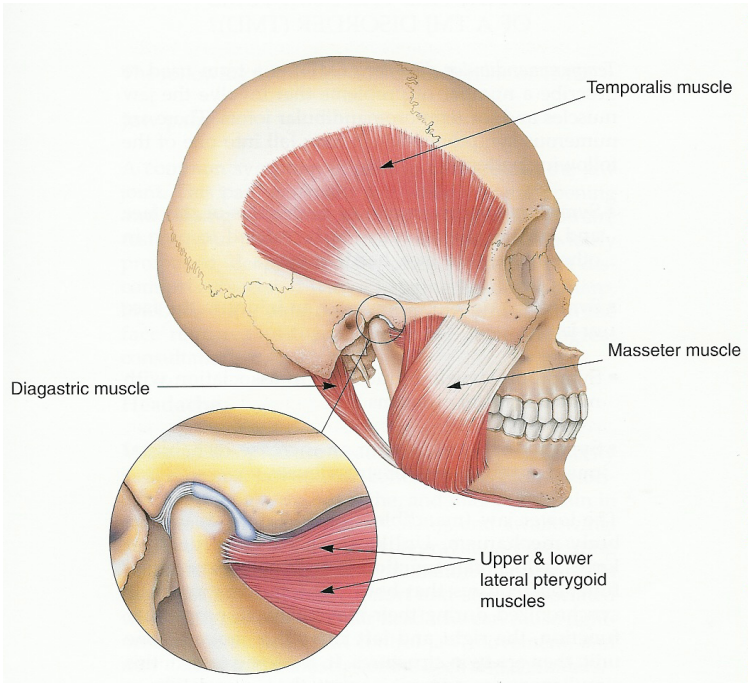

Image A: Anatomy masseter muscle. (Grays 2010)

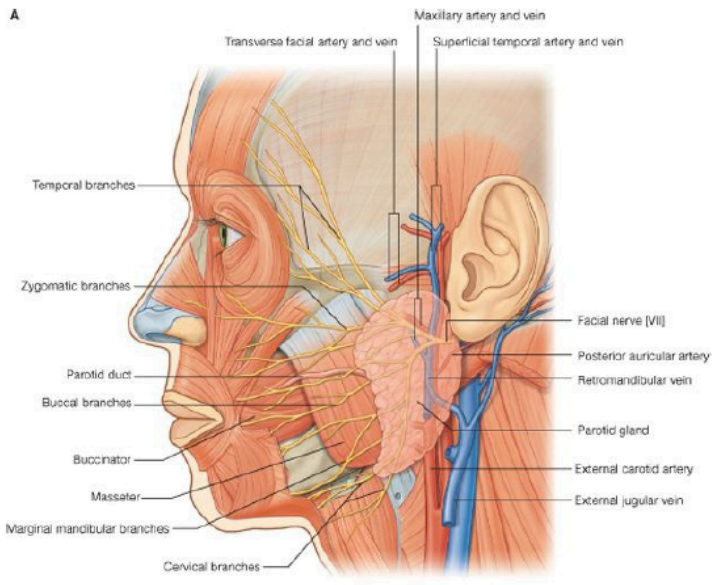

Image B: Anatomy masseter muscle and surrounding structures (Grays 2010)

Aetiology

Risk Factors

Sleep bruxism as well as teeth grinding are related to peripheral factors, for example, with negative dental occlusion due to tooth interference, central or pathophysiological causes and psychosocial influences such as stress or anxiety (Shetty S. 2010). Mostly, bruxism occurs during sleep, especially during the 'arousal phase' or REM (rapid eye movement) phase. During this phase the transition between deep and lighter sleep occurs and is associated with an increased heart and respiratory rate, as well as amplified muscle activity. In a related study, it was revealed that in 86% of bruxers, involuntary leg movement was associated with bruxing, substantiating the notion that the two processes are related (Macaluso 1998). Risk factors of bruxism include stimulating substances like caffeine, tobacco, heavy drinking, personality type where anxiety and stress sensitivity is common and other sleeping disorders such as snoring or sleep apnoea (Lavigne G. J. 2008).

Central or Pathophysiological Factors

Recently, research has shown that disturbances in central neurotransmitter system might contribute to the etiology of the bruxism. The central idea is that there is involvement of an immediate as well as an indirect pathway of the basal ganglion. The basal ganglion is a collection of five subcortical nuclei, which are partially responsible for the dexterity of movements. These subcortical nuclei can be disturbed in a patient suffering from bruxism. The output of the direct pathway travels straight from the stratum to the thalamus. At this point afferent signals are projected to the cerebral cortex. As for the indirect pathway, the signals primarily pass through several different nuclei prior to reaching the thalamus. If a difference occurs among the direct and indirect pathway a movement disorder can result. An example of such a disorder is Parkinson's disease. Bruxism patients may show an imbalance in both of the pathways rather than one or the other (Shetty S. 2010).

Peripheral Factors

Primarily, multiple occlusal factors were hypothesized to be contributing factors in bruxism as suggested in a study with self-reporting bruxism in children. Therefore, the managing of bruxism should include coordination between the maximum intercuspation and centric relation, yet recent studies agree on the fact that there is either no or a minimum affiliation between bruxing and the occlusal factors in adults. In a review by Manfredini (2004) the conclusion was made that is not sufficient data and a shortage of methodological

complete reports to definitely rule out the significance of occlusal factors in the etiology of bruxism (Shetty S. 2010).

Psychosocial Factors

Although there have been a variety of studies published concerning psychosocial factors when it comes to the etiology of bruxism, not one conclusively describes the nature of these factors because of a lack in large scale longitudinal trials. It has been noted in literature that bruxers differ from healthy individuals as they show signs of depression, hostility and sensitivity towards stress factors. Also, bruxing children are known to show more signs of anxiousness in comparison to children of the same age that do not brux, yet bruxing often stops during adolescence.

Daytime teeth clenching can be explained in terms of experienced, in other words, when one experiences stress they clench their teeth. Yet experienced stress and anticipated stress were shown not to be related to sleep bruxism in a study where ambulatory devices were used to record clenching activity (Van Selms 2004). Several studies show the growing possibility and relationship between a variety of psychosocial factors yet none of these have found to be conclusive (Shetty S. 2010)

Diagnosis of Bruxism

When diagnosing bruxism the first step is to identify 'parafunctions' in the patient when they are awake in combination with personal questions about how they experience the parafunctions and the visual observation of the patient's behavior. If there is suspicion of the disorder it is suggested to use home video to identify the unusual movements, as these unnatural movements might not show during a general dental exam. Generally, the most uncharacteristic movements are seen during the daily routine of a patient, and although it might seem practical to use an electromyogram (EMG) during this period, it is very difficult to discriminate the results between typical, speaking and chewing, and atypical orofacial movements. Oromandibular movements are considered to be unusual when they appear to be, too intense, frequent and interfere with typical movements such as speaking. A variety of movement disorders that commonly found in the oromandibular area are, uncontrolled mastication like movements, oromandibular myoclonus, excessive swallowing and Tourette's syndrome. All of the mentioned movements can be diagnosed or distinguished based on three points. Firstly, the observation of a parent or partner noting the sounds or motor activity, secondly clinical examination of tooth wear and jaw muscle hypertrophy and finally polygraphic and audio video recording of muscle activity with at an EMG. The golden standard in assessing sleep movement disorders

remains audio video and polygraphic recordings combined. This is due to the fact that it allows a more reliable as well as numerical evaluation of oromandibular actions. However, laboratory recording is a pricey and time costly manner of research in relation to data analysis as well as the fact that it is not very illustrative of how a patient sleeps in their natural environment. (Lavigne G. J. 2008)

Consequences of Bruxism

The results of sleep bruxism involve dental damage, temporomandibular joint and muscle pain or jaw lock. Jaw lock, better known as trimus, this is the term given to patients who show limited opening, which is usually caused by spasms of the masseter muscles. Also, 65% of bruxing patients report temporal headaches. Cheek biting is also a common consequence and is at its worse when patients suffer from xerostomia, or dry mouth. Temporomandibular disorders or chronic myofascial soreness in masticatory muscles may occur as well as hypertrophy of the masseter muscle. See image C. (Lavigne G. J. 2008). Finally, rather than medical problems, partner relationship problems can also be a result of bruxism as grinding sounds lead to partners to sleep separately as the noise can disturb the room partners, also patients might prefer to sleep alone because they are embarrassed by the sounds they make unconsciously.

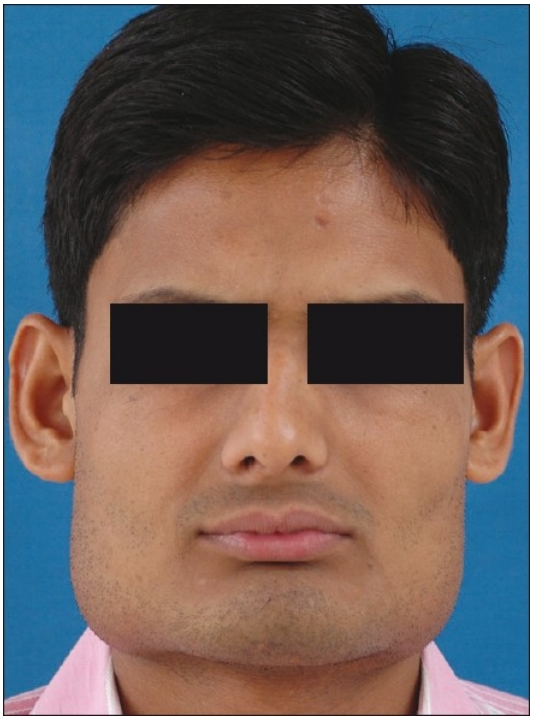

Image C – Male with Hypertrophic Masseter muscles (National Journal of *Maxilliofacial* Surgery)

Clinical use for Botulinum Toxin Type A

Botulinum toxin type A (BTX type A), is a substance that is synthesized by the organisms *Clostridium botulinum*, *Clostridium baratii*, and *Clostridium butyricum*, and is purified from culture via dialysis and acid precipitation to become the functional toxin used for treatments. The toxin preferentially performs on peripheral cholinergic nerve endings blocking acetylcholine release (see image F). Botulinum is known as agent that can treat disease yet the toxin can also cause the disease botulism (Simpson 2004). The effects of BTX type A begin forty-eight hours after the injection. To fully understand the process of BTX type A, it is important to first look at how the acetylcholine receptor works. The innervation of skeletal muscles comes from motor neurons whose cell bodies are in the spinal cord or in the brain stem. The axons of motor neurons travel from the central nervous system to the anterior spinal roots forming peripheral nerves. These then divide within the skeletal muscles creating terminals, which are connected to multiple striated muscle fibers. This forms the neuromuscular synapses. A motor unit is formed when a group of striated muscle fibers are innervated by one single motor neuron. When an action potential originating in the central nervous stem is released it runs down the motor neuron to the skeletal muscle fibers. When the action potential reaches the threshold it causes the motor neuron terminal to depolarize which allows the acetylcholine to be released into the neuromuscular synaptic cleft, this causes the Ca_{2+} concentration to increase. The release of acetylcholine from the cytosol is medicated and regulated by exocytosis. This involves multiple proteins that are collectively known as SNAREs (soluble N-ethylmaleimide–sensitive factor attachment protein receptors). As soon as the acetylcholine reaches the postsynaptic muscle membrane it is able to bind to a nicotinic cholinergic receptor, which in turn allows the opening of a transmembrane channel. This allows the entry of sodium ions into the muscle fibers and followed by the out flow of potassium. Due to the decrease in membrane potential within the muscle fiber, an endplate potential is generated. As soon as the endplate potential reaches the necessary threshold, the action potential is created in the muscle, causing it to contract. See image E.

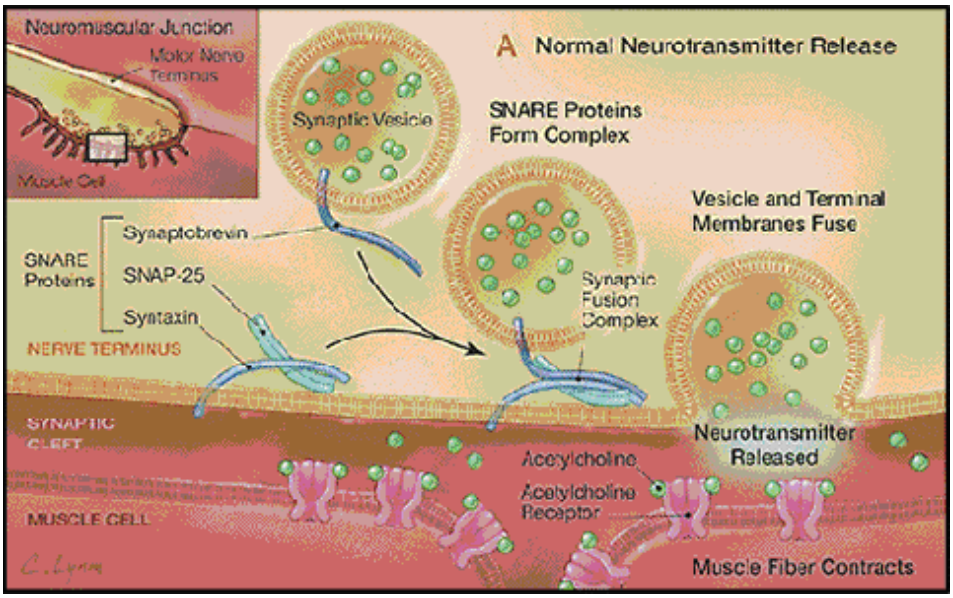

Image E – Normal Neurotransmitter Release - (Medical Device Source 2012)

BTX Type A is known to function through several steps. The initial step is the binding of Botulinum Toxin Type A to ecto-acceptors on the nerve terminal. This process is followed by an acceptor-mediated internalization of the molecule, translocation to the cytosol and finally the inhibition of Ca_2+ dependent neurotransmitter release. To be able to bind to the presynaptic acceptor, the C -terminal section of the heavy chain, which has the molecular weight of about 100 kDa, is necessary. The light chain on the other hand has molecular weight of 50 kDa. The toxin can then be internalized though receptor-mediated endocytosis up to the point that it is fully enclosed inside a vesicle. By now the functioning moiety can travel across the vesicle wall and the protease of the light chain can successfully cleave the protein that is necessary for the fusion of the vesicle and thus the release of acetylcholine. Summarizing, BTX type A, which has been taken up into the nerve terminals, is able to cleave the SNARE proteins. This then prevents the formation of the active merging complex and there by blocks the release of acetylcholine. (Medical Device Source 2012)

Clinically, the effects of BTX type A injections become significant between five and ten days. However muscle function restores over the next three to five months as the muscle progressively acquires new receptor sites and eventually can actively contract the muscle again. (Brannon H. 2008)

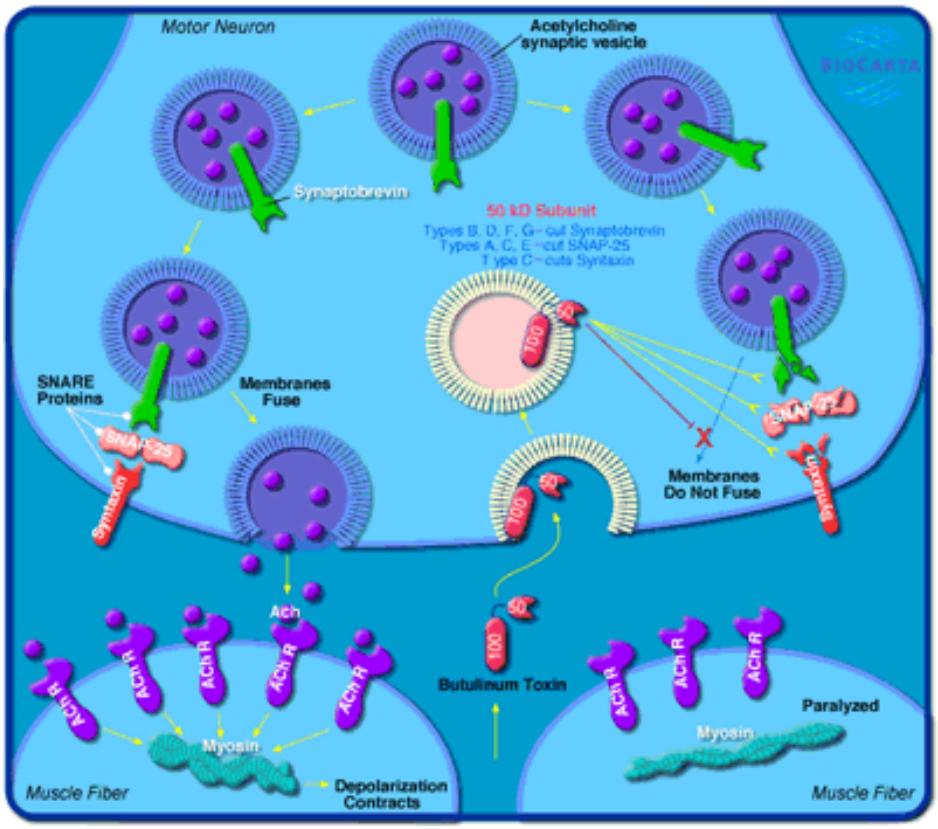

Image F – BTX type A blocking acetylcholine - (Medical Device Source 2012)

Currently, BTX type A is approved and is successfully used in the treatment of a variety of conditions that involve atypical and abnormal muscle spasms such as torticollis (neck twisting), hemifacial spasms (facial jerking), blepharospasm (eye spasms), chronic or frequent headaches, muscle rigidity, and in esthetic medicine.

Over the past decades it has become of increased importance to study the pharmacological management of bruxism. Drugs that effectively have a paralysing effect on the muscles have become increasingly popular in treatment of severe bruxism in cases such as coma, brain injury, amphetamine abuse, Huntington's disease and autism. Due to the fact that bruxism is caused by involuntary spasms of the masseter muscles, botulinum toxin has been tried, initial results showing that it is safe and effective. The BTX type A injections last up to sixteen weeks before the muscles start to regain their activity. In the treatment of bruxism they should be repeated after about three months (Ondo W. G. 2010).

Mouth guards

Mouth guards in the treatment of bruxism works by separating the teeth and thus preventing grinding. One can note if they are still grinding by observing the wear of the guard. Mouth guards generally take time to get used to but do effectively stop the habit of clenching. There is a significant reduction in face and jaw aching as well as a reduced likelihood dental damage. Mostly, patients only need to wear the mouth guard during the night yet if the clenching and grinding becomes an intractable habit they may need to wear it continually.

Mouth guards versus Botulinum Toxin Type A

Although it may seem that mouth guards are less invasive, they do require patient compliance. Also the discomfort of wearing a mouth guard can primarily lead to uncomfortable sleep. If the patients forget to regularly wear the mouth guard then the entire treatment would be ineffective. Besides this, mouth guards are generally an effective method in stopping the habitual mouth grinding but not in all cases. If the bruxing originates from spastic movements and not habitual movements the mouth guard will not stop the continual clenching but may relieve the damage to the teeth and possible Temporomandibular disorders. We can hereby conclude that this method does not per se work for all patients suffering from bruxism

BTX type A is a clear-cut manner in which muscle tensing is inhibited. Several small injections into the mandibular muscle of the jaw guarantee a stop to the clenching of the muscle and thus inhibit the bruxism activity. Also there is no need for patient compliance as in the case of mouth guards, making the treatment effective and easy. Bruxism can also cause an esthetically unpleasing size of the jaw, which is also easily treated by the use of BTX type A. BTX type A works in every patient, so also in patients who brux due to spastic movements rather than habitual movements. Finally it has been proven to be safe to use.

Conclusion

Although bruxism may seem as a quite simple disorder, there are many layers to it. It is a disorder that can take years to develop and if not paid attention to and lead to severe problems such as damage to the jaw joint. It is notable that some of the risk factors such as smoking and amphetamine use can be avoided yet a stressful life style or a disposition towards stress sensitivity are also factors to look out for. Causes of bruxism are not completely conclusive yet can be linked back to pathophysiological factors, central neural factors, peripheral neuromuscular factors and psychosocial factors. Bruxism can be managed in several ways, for example with BTX type A injections. The light chain botulinum toxin type A can actively cleave the SNARE proteins that are responsible for the fusion of the vesicle that allows the release of acetylcholine causing muscle contraction. Another way to manage bruxism is with the use of mouth guard, yet this can be uncomfortable and patient compliance is necessary. BTX type A injections on the other hand last up to four months and have other benefits, like the fact that it doesn't need patient compliance. One can conclude that there still needs to be a lot more research done in the field of bruxism to find the underlying cause of the disorder.

Works Cited

- Brannon, H. "The Science of Botox." *About.com Dermatology*. About.com, 3 Oct. 2008. Web. 03 July 2013.
- DrugBank: Botulinum Toxin Type A (DB00083)." *DrugBank: Botulinum Toxin Type A (DB00083)*. Genome Canada, 13 June 2005. Web. 03 July 2013.
- El Maaytah M, Jerjes W, Swinson B, and Hopper C. "Bruxism Secondary to Brain Injury Treated with Botulinum Toxin-A: A Case Report." Head and Face Medicine 2 (2006): n. pag. Head & Face Medicine. 23 Nov. 2006. Web. 15 Mar. 2013
- Gray, H., R. L. Drake, W. Vogl, A. W. M. Mitchell, R. Tibbitts, and P. Richardson. *Gray's Anatomy for Students*. Philadelphia: Elsevier/Churchill Livingstone, 2010. Print.
- Lavigne, GJ, S. Khoury, K. Raphael, S. Yamaguchi, and S. Abe. "Bruxism Physiology and Pathology: An Overview for Clinicians." Journal of Oral Rehabilitation 35.7 (2010): 476-94. Pub Med. Web. 15 Mar. 2013.
- Macaluso GM et al (1998) Sleep bruxism is an disorder related to periodic arousals of sleep. J Dent Res 77:565
- *Male With Hypertrophic Masseter Muscles*. Digital image. *National Journal of Maxilliofacial Surgery*. N.p., 10 Oct. 2011. Web. 3 July 2013.
- Manfredini D, Cantini E, Romagnoli M, Bosco M (2003) Prevalence of bruxism in patients with different research diagnostic criteria for temporomandibular disorders (RDC/TMD) diagnoses. Cranio 21:279–285
- Medical Service Source. *Botox Blocking Acetylcholine*. Digital image. *Medical Device Source*. Medical Service Source, 2012. Web. 3 July 2013.
- Medical Service Source. "Botox." *Medical Device Source*. Medical Service Source, 2012. Web. 03 July 2013.
- Medical Service Source. *Normal Neurotransmitter Release*. Digital image. *Medical Device Source*. Medical Service Source, 2013. Web. 3 July 2013.

- Mayo Clinic., Staff"Bruxism." *Mayo Clinic*. Mayo Foundation for Medical Education and Research, 19 May 2011. Web. 03 July 2013.
- Santamato A, Panza F, Di Venere D, Solfrizzi V, Pierto Fioreh, and Ranieri M . "Help Journal List J Chiropr Med V.9(3); Sep 2010 PMC3188369 Logo of Jchiromed J Chiropr Med. 2010 September; 9(3): 132–137. Doi: 10.1016/j.jcm.2010.04.004 PMCID: PMC3188369 Effectiveness of Botulinum Toxin Type A Treatment of Neck Pain Related to Nocturnal Bruxism: A Case Report." Journal of Chiropractic Medicine 9 (2010): 132-37. Print.
- Shetty S, Pitti V, Babu S, Kumar G. P. S, and. Deepthi B. C. "Bruxism: A Literature Review." Indian Prosthodontic Society 10 (2011): 141-48. PG. Web. 15 Mar. 2013.
- Simpson, L L. "Identification of the Major Steps in Botulinum Toxin Action." Annu Rev Pharmacol Toxicol 44 (2004): n. pag. Pub Med. Web. 15 Mar. 2013.
- Ondo, W G. *Study of the Safety and Efficacy of Botox in Bruxism*. N.p.: n.p., n.d. *Clinical Trials*. Baylor College of Medicine, May 2010. Web. 03 July 2013.
- Van Selms MKA, Lobbezoo F, Wicks DJ, Hamburger HL, NaeijeM (2004) Craniomandibular pain, oral stress in a longitudinal case study. J Oral Rehabil 31:738–745