Essential Palatal and Nostril Myoclonus Patient Relieved by Disclusion Time Reduction therapy: A Case Report.

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Abstract

Objective: Essential Palatal Myoclonus and Perioral Myoclonus (POM) are rare soft palate conditions which may present idioopathically or with structural brain epilepsies. Palatal Myoclonus is characterized by uncontrolled, rhythmic, jerky movements of the soft palate, which can be accompanied by other ear, nose, and throat symptoms that appear in the muscles innervated by the brainstem. A particularly annoying symptom is a rhythmic clicking sound in the ear, resultant from the uncontrolled palate movements repeatedly opening and closing the Eustachian tube. Alternatively, Orofacial Buccal Dystonia presents as focal, sustained spasms of the masticatory facial or lingual muscles, and of the nostrils. Both of these related spastic muscle conditions can best be diagnosed with a proper understanding of the functional orofacial neuroanatomy that is responsible for the clinical signs and symptoms. This Clinical Report details a rare case of Palatal Myoclonus with inner ear clicking that was associated with both Orofacial Buccal Dystonia and Nostril Myoclonus. The patient underwent an objective T-Scan 10/BioEMG III functional occlusal analysis and was diagnosed with Occluso-muscular Disorder, which is a subset of Temporomandibular Disorders (TMD). By objectively calibrating the patient’s occlusion with the computer-guided Immediate Complete Anterior Guidance Development (ICAGD) coronoplasty, both the Palatal and Nostril Myoclonus were successfully resolved along with the sustained spasms of the masticatory facial muscles.

Key Words: Palatal Myoclonus, Perioral Myoclonus, Nostril Myoclonus, Disclusion Time Reduction, Occlusion Time, Immediate Complete Anterior Guidance Development (ICAGD) Coronoplasty
Introduction:

Palatal Myoclonus is a movement disorder in which there are rhythmic, involuntary jerky movements of the soft palate. Etiologically, Palatal Myoclonus has been classified into two distinct forms; 1) Symptomatic Palatal Myoclonus, a condition that is secondary to identifiable brainstem or cerebellar disease and 2) Objective Tinnitus or Essential Palatal Myoclonus, a condition that presents with clicking tinnitus in the absence of a brain lesion.

Objective Tinnitus can be of muscle origin, vascular origin, and from a patulous Eustachian tube. Further, patients who experience symptomatic Palatal Myoclonus often undergo a pathologic, hypertrophic degeneration of the inferior olive and dentate nucleus, which then initiates rhythmic, spontaneous, synchronized discharges within the inferior olive. Through this degenerative mechanism, the brainstem becomes an “oscillator” that repeatedly contracts the soft palate.

In contrast, there is no known etiology for Essential Palatal Myoclonus. A suggested, but not yet proven pathophysiologic hypothesis, is that a spontaneous oscillation develops in the parts of the brainstem that relate to palate motor function (the nucleus ambiguous, the elevator palatini, facial nucleus, the median and paramedian muscles, and the hypoglossal nucleus, the palatopharyngeal and palatoglossus muscles). Palatal Myoclonus should be distinguished from Middle Ear Myoclonus, where the tinnitus sound is a buzzing caused by high-frequency tympanic membrane movement, rather than palatal tremors inducing rapid Eustachian tube opening and closing.

Orofacial Dystonia is a focal dystonia with sustained spasms of the masticatory, facial, or lingual muscles which affects more women than men between ages 31 - 58 years. Neck and limb muscles spasms may accompany the facial dystonia and in most cases the cause of spasm is unknown (idiopathic or primary), and the disease condition arose spontaneously. Suggested possible Orofacial Dystonia etiologies are drug-induced, are peripheral disease-induced, are associated with post-anoxia, or are associated with a neurodegenerative disorder, or a head injury.

The current lack of knowledge of the exact pathophysiology of both Dystonia and Palatal Myoclonus make them both difficult to treat, pharmacologically. A wide variety of medications have been tried including cholinergic, benzo diazepam, anti-parkinsonism drugs, anticonvulsants, muscle relaxants, 5-hydroxytryptophan, levodopa and lithium. A more accepted pharmacologic therapy for both Palatal Myoclonus and Orofacial Dystonia attempts to control neurologic transmission in parts of the brainstem using Botulinum Toxin (BTX-A), which blocks somatic neurologic input and output that incites the involuntary muscle movements. Botulinum Toxin inhibits the calcium-mediated release of acetylcholine into the synaptic junction, resulting in local chemical denervation and loss of neuronal activity in the targeted organ. Of note is that BTX-A has been used in the treatment of many different abnormal movement disorders.

Surgical options also exist for Palatal Myoclonus, which includes Eustachian tube obliteration with subsequent ventilation tube placement, with/without dissecting the palatal muscles. Detaching the involved muscles has also been attempted to treat Orofacial Dystonia. And counseling and psychiatric care often play a role in the management of these rare spastic muscle disorder patients.

Prolonged in time, excursive movement occlusal interferences and occlusal surface friction are contributory etiological factors for Occluso-muscular pain and symptoms often observed in TMD patients. A commonly observed TMD symptom that results from these prolonged interferences is masseter muscle spasm, which has been successfully treated with the measurement-driven, computer-guided occlusal adjustment procedure known as Immediate Complete Anterior Guidance Development (ICAGD). ICAGD’s primary therapeutic goals are to measurably decrease the Disclusion Time (DT) required for all molars and premolars to disclude from each other in less than 0.5 seconds during their right and left mandibular excursions commenced from the Maximum Intercuspal Position (MIP).

This coronoplasty utilizes the T-Scan 10/BioEMG III synchronized technologies (T-Scan version 10, Tekscan, Inc. S. Boston, MA, USA; BioEMG III, Bioresearch Assoc., Milwaukee, WI, USA) record real-time force movies of mandibular excursive movements, with those same movement’s associated masticatory muscle contraction patterns. The synchronized programs measure and verify that specific MIP closure and excursive movement time-duration numerical endpoints have been achieved to qualify that the ICAGD occlusal adjustments were accurately accomplished. Properly performed ICAGD has been shown to markedly reduce muscle hyperactivity levels, where many different authors using ICAGD have reported rapid muscular TMD symptom resolutions. And an important patient benefit of undergoing ICAGD is that it successfully treats Occluso-muscular
TMD symptoms without requiring the treated patient to be repositioned out of MIP, wear a splint, or be pretreated with a deprogrammer.\textsuperscript{17-34}

The beauty of this computer-guided treatment approach is that it lessens hyperactive muscles from within the Central Nervous System (CNS) by controlling the volume and time-duration of molar and premolar pulpal flexure and the Periodontal Ligament (PDL) mechanoreceptor compressions, which synapse directly with effector motor fibers that contract the four masticatory muscles and the muscles associated with swallowing.\textsuperscript{31, 35} The pulpal flexure duration and PDL compression durations equal to the Disclusion Time duration of that same excursion\textsuperscript{21}, so the more time the excursive interferences contact, the longer the pulpal fibers and periodontal fibers contract the muscles innervated by the Trigeminal Motor Nucleus.\textsuperscript{23, 26, 27} ICAGD reduces the length of time that posterior occlusal surfaces contact excursively, which in turn, reduces the duration that pulps flex and PDL mechanoreceptors are compressed. This effectively interrupts muscle hypercontraction, allowing for muscle re-oxygenation, muscle fiber recovery, and then muscle spasm resolution.

Recently, a rare case of Orofacial Dystonia presented to the Raja Rajeshwari Dental College, Rajiv Gandhi University of Health Sciences. The patient complained of an annoying clicking sound in the ear (tinnitus), being diagnosed with Essential Palatal Myoclonus with Nostril Myoclonus, and associated Orofacial Buccal Dystonia. To date, this appears to be the first Case Report of an orofacial dystonia that predominantly presented as Palatal Myoclonus. After completing the patient’s technology-based diagnostic workup (detailed below), a significant pathologic occlusal component was detected. This was illustrated by a large right-side-to-left-side MIP occlusal force percentage imbalance, and the presence of long Disclusion Time and masticatory muscle excursive hyperactivity in his excursive movements.

The T-Scan recordings suggested this patient was suffering from an occlusal problem that, because the other involved structures are innervated by branches of the Trigeminal Nerve (CN V), might be contributory to the disabling symptoms in his palate, ear, and orofacial area. As such, Disclusion Time Reduction therapy with the ICAGD coronoplasty was recommended as treatment for this rare myoclonus patient. Once his prolonged pretreatment Disclusion Times were reduced to short Disclusion Time, both the Palatal and Nasal Myoclonus resolved, as did most of the patient’s Orofacial Buccal Dystonia symptoms.

**Case Report History**

A 30-year-old male presented with a 6-year history of persistent clicking tinnitus on right side, right side facial pain, continuous tremors of soft palate and both nostrils, which over its 6-year development, also spread into the right neck area. The patient’s tinnitus and involuntary palatal movement developed following a cold attack. The right-sided facial, buccal, and nostril involuntary dystonic tremors were noticeable to other people and they affected the patient’s speech. The clicking and involuntary facial muscle contractions prompted the patient to visit several dental, otolaryngology, and neurology clinics. But over time, both the tinnitus and painful dystonic spasms grew more aggravated, and was worsened by everyday life stressful events. Adding to the patient’s stress was that after 6 years of seeking help, a diagnosis had still not been made. The patient felt rejected, sad, depressed, and grew more anxious the longer the painful symptoms remained undiagnosed. Frustration also worsened as he tried different treatment modalities (herbal medicine, acupuncture and massage), that brought no marked relief nor slowed the progression of the symptom worsening.

![Figure 1. Pre-treatment CBCT Image obtained in December of 2019, showing degenerative changes in both TM Joints in the transverse view. In the sagittal view, there is visible narrowing of upper and middle airway.](image-url)
As there were specific locations of the spastic painful symptoms, the palate (intraoral) and facial (extraoral) areas were thoroughly examined but presented structurally within normal limits. However, a bilateral symmetric contraction of the anterior margin of the soft palate occurred while both nostrils contracted, as synchronous patient clicking tinnitus was observed in the right ear. The patient reported that he also felt involuntary movements of the muscles lateral to the nose and down into the anterior neck. These simultaneously occurring physical manifestations confirmed the suspicion that the patient was experiencing Palatal Myoclonus with Orofacial Buccal Dystonia. Diagnostic imaging with Cone Beam Computed Tomography (CBCT), see Figure 1, and Brain MRI did not reveal any abnormal brain or brainstem lesions. Consultation with the Neurology department ruled out other neurologic disorders due to the absence of any other accompanying neurologic deficits. The structural and functional neurologic health when combined with the patient report of symptoms, led to a diagnosis of Essential Palatal Myoclonus and Nostril Myoclonus associated with Orofacial Buccal Dystonia. The CBCT imaging did detect subchondral sclerosis of both mandibular condyles. This finding required the patient undergo a Temporomandibular Joint Vibration Analysis (JVA, Bioresearch Assoc., Milwaukee, WI, USA) (Figures 2a and 2b), that was followed by a T-Scan10/BioEMG III functional occlusal analysis of his occlusal force distribution, his excursive movement contact patterns, and the masticatory muscular contraction patterns related to that occlusal function (Figures 3, 4 and 5).

**Figure 2a.** Joint Vibration Analysis (JVA) showed a normal (54 mm) range of motion and low amplitude low frequency waveforms indicative of good dynamic joint function in fully-adapted and most likely stable TM Joints.

**Figure 2b.** The JVA’s FFT distribution, which showed little energy above 300 Hertz, confirming that the degenerative hard tissue changes seen in Figure 1 were most likely stable without current active disease (Piper 5b).
The patient was prepared with EMG electrodes placed on the bilateral masseter and temporalis muscles, which were connected to the BioEMG III amplifier. Next, the patient intercuspidated firmly into the T-Scan HD sensor to make a 3-closure Multi-bite recording. This was followed by a left excursive movement recording (Figure 3), where the patient again intercuspidated firmly into the T-Scan sensor, then held his intercuspidated MIP (maximum intercuspal) for 1-3 seconds, and then glided to the left out to the tips of his opposing canine teeth. A right excursive recording was also made following the same procedure (Figures 4 and 5).

**Figure 3.** The Pre-treatment Multi Bite recording showed that the patient’s ability to maintain intercuspation at full force was significantly compromised in 2 of the 3 clenches. This was a marker for muscle fatigue that was coming from the occlusion.

**Figure 4.** The Pre-treatment Left Lateral recording where the Disclusion Time was prolonged at 1.45 seconds, and the Time to Muscle Shut Down after the excursion began at C was very long = 3+ sec.
Diagnostic TMJ and Occlusion Functional Examination

The JVA report confirmed that although there was an internal disc derangement, the patient had bilaterally quiet TMJs with a normal range of motion at 54 mm (Figure 2a) and minimal high frequency vibration content. This did not indicate an active degenerative joint disease (Figure 2b), suggesting adapted and most likely stable TMJs. Additionally, there were prolonged right and left excursive Disclusion Times; the left disclusion time was 1.45 seconds, the right was 2.40 seconds, (Figures 4 and 5), both of which far exceeded the known physiologic range of less than 0.5 seconds. Of note was that the Time to Muscle Shut Down of the working temporalis and masseter muscles (Figures 4 and 5) ranged between 4 to 5 seconds, which is far too long to be physiologic. This prolonged muscle firing is known to cause lactic acid ischemia, which in this patient induced the facial pain and muscular spasms the patient had experienced for years. It was explained to the patient that his Disclusion Times were too long, and were causing his excursive muscle hyperactivity (Figures 4 and 5 in the upper right EMG pane to the right of Line C), and his ongoing muscular symptoms. He was also told that his muscular pains could be reduced or eliminated if he elected to have his Disclusion Times reduced with the ICAGD coronoplasty. What remained as a question was whether his Essential Palatal Myoclonus and Nostril Myoclonus were also somehow neurologically related to his long Disclusion Times.

After obtaining the patient’s informed consent, the ICAGD enameloplasty was performed on his right and left excursive movements as previously described to remove the prolonged occlusal surface friction and reduce the DTs to within physiologic durations. Post ICAGD, the corrected closure into MIP produced an Occlusion Time of 0.20 seconds (Figure 6), the left excursive DT was 0.31 seconds (Figure 7) and the right excursive DT was 0.23 seconds (Figure 8). When comparing the pretreatment excursive EMG hyperactivity (Figures 4 and 5) with the post ICAGD excursive EMG data, the excursive hyperactivity in both treated excursions was markedly lessened following ICAGD (Figures 7 and 8 in the upper right pane, to the right of Line C).

The Time to Muscle Shut Down of both working masseter and temporalis muscles was drastically shortened after the ICAGD corrections. The reason that ICAGD was effective in treating the TMD symptoms is that ICAGD shortens the contraction times of the involved muscles, thereby reducing lactic acid buildup and ischemia, while enhancing the metabolism with fiber re-oxygenation and muscle healing. For the first time in 6 years, now with short Disclusion Time, the patient’s Palatal and Nasal Myoclonus, and most of his Orofacial Buccal Dystonia symptoms, quickly resolved.
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Figure 6. The Post ICAGD Multi Bite showed the patient was able to hold their clenched intercuspation in all 3 clences, which he could not do pre-treatment (Figure 5). There are higher amplitudes of the clench EMG data, and there are taller and flatter force curves in the T-Scan’s Total Force Line that no longer drop off from muscle weakness. The MIP contacts are highly balanced with the COF centered.

Figure 7. The Post ICAGD Left Lateral recording with short Disclusion Time = 0.39 second, and markedly less muscle contractions, and a much shorter time to muscle shut down after C compared to Figure 6. The Time to muscle shut down after ICAGD = 0.35 second.

Figure 8. Post ICAGD Treatment Right Lateral recording with short Disclusion Time = 0.36 seconds, and markedly less muscle contractions and shorter time to muscle shut down after C compared to Figure 7. The Time to muscle shut down after ICAGD = 0.2 sec.
Discussion

Since muscular TMD symptoms respond well to computer-guided occlusal adjustment therapy, this patient was advised to undergo the ICAGD treatment. The patient reported this was the first time a non-pharmacological treatment method was used and it was the only treatment the patient benefited from. Both the patient’s therapeutic response and the outcome of this Palatal Myoclonus Case Report align very closely with the numerous ICAGD studies and Clinical Reports that have described the rapid physiologic healing effect that Disclosure Time Reduction offers myogenous TMD sufferers. Although Treating Occluso-muscle dysfunction with ICAGD began in 1991 and therefore is not new, this is the first Case Report where increased Disclosure Times were found to be hyper-contracting and spasming the soft palate, and that reduction of the Disclosure Time to less than 0.5 seconds reduced the soft palate spasms, and their related Eustachian tube opening and closing.

Palatal Myoclonus is classified as an objective tinnitus when the symptoms arise from a connection lesion between the dentate nucleus, the red nucleus, and the inferior olivary nuclei (Guillain-Mollaret triangle). Thus, the “Essential” classification is denoted when there is no identifiable brain lesion, but contractions of the levator veli palatini and tensor veli palatini muscles cause rapid uncontrollable opening and closing of the Eustachian tube that produces an audible clicking tinnitus (that was clearly audible in this patient). Additionally, Orofacial Dystonia, which is a part of Oromandibular Dystonia, has been reported by both neurologists and dentists.

The diagnosis of dystonia is accomplished clinically to distinguish between the different focal types of dystonia (mandibular dystonia, orofacial mandibular dystonia, tongue dystonia, and cranial dystonia). Although the apparent trigger for this onset of symptoms was a cold attack, the explanation how the longstanding Palatal Myoclonus responded to an occlusal therapy lies in the synaptic neuroanatomy the nerve fibers in posterior teeth, pulp, and the periodontal ligaments have within the Trigeminal Motor Nucleus. The molar and premolar pulps and periodontal ligament mechanoreceptors are the only afferent human Peripheral Nerves that do not synapse initially outside the Central Nervous System. Instead, the pulp and PDL mechanoreceptors enter the CNS directly via the Mesencephalic Nucleus, and without synapsing there, travel on further to the Trigeminal Motor Nucleus bilaterally, while also traveling on to the Reticular Formation.

The Reticular Formation is the major brain center that controls functions like swallowing, waking, posture, and breathing. Within the Trigeminal Motor Nucleus, the pulpal and PDL mechanoreceptor fibers make their first synapse with the efferent motor fibers to the 4 muscles of mastication, the tensor tympani, the tensor veli palatini, the mylohyoid, and the anterior belly of the digastric muscles, which all together comprise the Swallow Mechanism muscles. This direct afferent synapse between the prolonged contact durations of the posterior teeth and the tensor veli platini muscle, is what triggered the soft palate into its rhythmic uncontrolled contractions that repeatedly opened up the patient’s Eustachian tube. Once the Disclosure Time was shortened, the hypercontraction impulses coming from the pulp and PDL fibers were removed from the neurophysiology, thereby stopping the soft palate spasm.

An explanation for why this patient’s nostrils were also contracting, lies in the fact that the Facial Nerve (CN VII) uses some fibers of the Trigeminal Nerve (CN V) to reach muscles the Facial Nerve innervates. The afferent pulpal and periodontal ligament neurologic output resultant from the prolonged posterior tooth contact durations creates efferent output from the CNS that travels along the 3 branches of the Trigeminal Nerve. Some fibers of the maxillary division (V2) are used by CN VII such that the effferent neural output on V2 was transmitted to the accompanying CN VII fibers, that innervated the muscles of facial expression approximate to the nose. Prolonged Disclosure Time magnified the effferent contraction impulses in both volume and in time-duration, thereby spasming the muscles near the nose. In this patient, in the same way that short Disclosure Time reduced the masseter muscle contractions after ICAGD, so did the nostril twitch contractions abate.

In this presented case, the ICAGD adjustments interrupted the longstanding hypercontraction of the tensor veli palatini, thereby resolving the Essential Palatal Myoclonus, and opening the patient’s upper and middle airway visibly within the post treatment CBCT scans. All of these physiologic improvements resulted without the patient wearing an occlusal or sleep appliance, undergoing TENS, taking pain, muscle relaxant, or anti-inflammatory medications, or requiring trigger point and/or Botox injections.

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Figure 9a. General somatic afferent nerve pathway (in green) from molar and premolar pulp and PDL mechanoreceptors that passes through the Mesencephalic Nucleus (MesNU) to later synapse in the Trigeminal Motor Nucleus (TrMoNU) bilaterally, and also travel onto the Reticular Formation (reprinted with permission from Haines Neuroanatomy, 8ThEd., Lippincott, Williams, and Wilkins publishers, http://lww.com).
Figure 9b. Structures innervated by the Trigeminal Motor Nucleus (TrMoNU) in Figure 9a. These innervated muscles comprise the Swallow Mechanism muscles, and include the tensor veli palatini (reprinted with permission from Haines Neuroanatomy, 8th Ed., Lippincott, William and Wilkins publishers, http://lww.com).
Figure 10a. The post ICAGD CBCT Image obtained in February of 2020, showing improved TM Joint health from the occlusal changes to the Disclusion Time. In the sagittal plane, there is a visible widening of the upper and middle airway.

Figure 10b. Comparison Airway CT views with measurements from before (A & B) to after ICAGD (C & D). The upper and middle airway dimensions increased from ICAGD relaxing the pharynx without using a mandibular advancement or sleep appliance.
This Case Report illustrates the far reach of noxious output that emanates from the pulp and periodontal ligament afferents, which can be controlled external to the CNS by ICAGD. Properly performed ICAGD therefore has many advantages in the treatment of occlusally activated muscular symptoms when compared to; 1) the unmeasured occlusal equilibration involving Centric Relation manipulation,\(^2\)\(^,\)\(^3\) to the neuromuscular mandibular advancement that requires crowning many teeth after the Myobite is established and 3) to the many other commonly employed, symptom-focused, non-occlusal therapies such as “self-care” and cognitive behavioral therapy.

**Summary**

This Case Report describes an Occluso-muscle Disorder patient that had his left side to right side force imbalance corrected and his prolonged pretreatment Disclusion Time reduced to short Disclusion Time by undergoing the ICAGD enameloplasty. Shortly thereafter, the patient’s Essential Palatal Myoclonus and Nostril Myoclonus associated with Orofacial Buccal Dystonia produced tinnitus completely stopped, the involuntary nostril tremors completely resolved, the Orofacial Buccal Dystonia and pain had decreased to tolerable levels, and the patient’s airway increased. The neurologic stimulus coming from the occlusion was markedly reduced by ICAGD lessening the prolonged excursive posterior tooth contact timing durations. This occluso-neurologic approach directly impacted the physiology of patient’s swallowing muscles to eliminate the Essential Palatal Myoclonus.

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