

Bruxism Through the Eyes of A Wet-Fingered Dentist

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Introduction

Dentistry is fast changing and we all struggle to keep up. What I was taught about bruxism as a student in dental school in the late seventies and what I taught at the faculty of dentistry had expiry dates.

As part of the forward planning for the NUS Edmund Tay Mai Hiong endowment fund, a questionnaire survey (n=74) was conducted to assess the current knowledge and attitudes of local dentists regarding sleep and airway issues in dentistry. Alarming, 45% of responders were unaware that most sleep bruxism (SB) episodes occurred as a consequence to sleep micro-arousals and, worse, 39% still believed erroneously that '*SB is an occlusal disorder*'. Only 35% (not necessarily those who answered the two previous questions correctly) volunteered that they 'felt confident' in managing patients with SB!

I have been in private practice limited to the specialty of prosthodontics since 1993. My three areas of special interests are: interdisciplinary full mouth occlusal rehabilitation, the management of chronic orofacial pain and temporomandibular disorders (TMD), and dental sleep medicine. What I have learnt from the latter two sectors have transformed my practice of prosthodontics, and I would like to share some of my clinical insights with you.

Consensus in the definition & diagnosis of bruxism

Bruxism is defined as "*a repetitive jaw-muscle activity characterized by clenching or grinding of teeth and/or by bracing or thrusting of the mandible. It has two distinct circadian manifestations: it can occur during sleep i.e. Sleep Bruxism, or during wakefulness i.e. Awake Bruxism.*" (Lobbezoo F et al 2013)

Many full-mouth prosthodontic reconstruction cases are the deleterious consequences of bruxism. Whenever I see any patient referred to me for the management of 'abnormal' tooth wear, I would always ask the following questions:

- *Is this wear functional or parafunctional?*
- *Is it historic or ongoing?*
- *If ongoing, is it occurring when the patient is awake, asleep or both?*
- *If during sleep, does it occur in Non Rapid Eye Movement (NREM) sleep only, Rapid eye movement (REM) sleep only or both?*
- *Are we dealing with a primary (idiopathic) sleep bruxism, secondary sleep bruxism, or a combination of the above?*

Bruxism is usually categorized as *possible, probable, putative or definite*. A diagnosis of 'definite' bruxism can be made only if there is polysomnographic (PSG) evidence, in addition to self-report (usually collected via questionnaires and/or patient history), and physical examination. Although the use of PSG for diagnosing bruxism may be useful in research, it is not routinely used in daily clinical dental practice.

Disadvantages of PSG in the diagnosis of Sleep Bruxism (SB) in daily practice

In diagnosing SB, a very specific electromyographic (EMG) surrogate called **rhythmic masticatory muscle activity (RMMA)** is studied. RMMA is defined as 3 masseter muscle bursts or contractions (phasic, tonic or mixed) within an episode in the absence of teeth grinding.

This type of attended sleep study requires sophisticated instrumentation, high levels of technical competence, is time-consuming and very expensive. Patients are made to sleep and are studied in a very unnatural setting with two belts around their chest and abdomen, and many recording electrodes on their face and body.

Bruxism Phenotypes

Dentists generally become involved only when there is observable occlusal interface and/or other stomatognathic damage (e.g. TM joint dysfunction, masticatory muscle symptomology). It is paramount to understand the subcategories of bruxism in order to tailor appropriate treatment for these patients.

(A) NREM Sleep Bruxism (SB)

In NREM sleep, most micro-arousals tend to occur in a structured and repetitive manner known as the cyclic alternating pattern (CAP). SB patients are thought to have a heightened responsiveness to sleep arousals. NREM SB is likely '*an extreme manifestation of a complex physiologic oromotor behavior*' that serves a homeostatic purpose for maintenance of oro-esophageal pH and lubrication as well as upper airway patency during sleep. Therefore, there is intensification of the rhythmic masticatory muscular activity (RMMA) in either frequency and/or amplitude in predisposed individuals. This is the same for those with hyper-arousal from other painful comorbidities, insomnia or fragmented sleep due to unresolved Upper Airway Resistance Syndrome (UARS) or Periodic Limb Movement Syndrome (PLMS). Fluctuations in central nervous system neurochemicals (e.g. dopamine) and drugs (e.g. clonidine) that influence sleep architecture by altering REM sleep onset (e.g. in depression) may also influence the occurrence of RMMA during sleep.

i. Implications 1: NREM SB in children

We used to reassure worried parents that 1 in 3 children typically stop their nocturnal tooth grinding when they reach their twelfth birthday. The observation was valid although previously we really didn't know the reason for it. Since the human airway only reaches its mature dimension around 12 years, whereas adenoid growth begins at 2.5 years and peaks at around 5 to 6 years, manifestations of NREM SB in children should therefore alert us that there are ongoing airway patency issues during sleep (e.g. adeno-tonsillar hypertrophy, chronic nasal congestion, allergic rhinitis). Hence sleep bruxism, snoring and restlessness in bed observed are likely the consequence of airway challenges during sleep. Therefore, these children should be referred to the paediatric sleep physician or ENT surgeon for further investigation.

ii. Implication 2: SB in patients with erosive wear

SB may also be a protective arousal-related response to stimulate saliva secretion, which enables the neutralization of oesophageal acid. NREM sleep bruxers with comorbid Obstructive Sleep Apnea (OSA) or Upper Airway Resistance Syndrome (UARS) who also present with severe dental attrition combined

with erosive wear should be screened for sleep-related gastro-esophageal reflux disorder (GERD) and laryngeal-pharyngeal reflux (LPR) because of the associated increase in negative intrathoracic pressure. If confirmed, these patients should be treated with proton pump inhibitors e.g. *Omeprazole* in addition to protective occlusal appliances worn during sleep. (Figure 1)



Courtesy of Edmund Toy Mai-Hiong Endowment

Figure 1: Phasic NREM SLEEP BRUXISM with comorbid UARS and GERD (i.e. Secondary SB)

iii. Implication 3: Choice of nocturnal occlusal devices

Interestingly, the conventional full occlusal coverage flat plane '**stabilization splints**' we were all taught in Dental School to fabricate for bruxism are now *contraindicated* in patients with sleep disordered breathing as they have been shown to actually worsen the sleep apnoea and cause respiratory disturbance!

(B) REM Sleep Bruxism

Only 10% of sleep bruxism occur in REM sleep. REM SB has an entirely different etiopathology from NREM SB and is possibly a subclinical manifestation of REM sleep behaviour disorder (RBD).

RMMA-SB occurring during REM has special clinical significance because of the general attenuation of protective reflexes during this phase of sleep. REM SB has previously been referred to as “**DESTRUCTIVE BRUXISM**”. About 1 in 4 patients referred to me for prosthodontic full mouth reconstruction for severe dental attrition exhibited REM SB confirmed by PSG. They were found to have more RMMA-SB episodes and more bruxing per min of REM sleep. They presented with significant morning symptomology (dental damage, odontalgia, TMJ arthralgia, masticatory muscle myalgia, delayed onset muscle soreness and/or transient headache). Without nocturnal occlusal splint protection, many properly osseointegrated implant-supported prostheses will fail in these patients.

During REM bruxism, recurrent microtrauma from unrestrained mandibular torquing caused by the vector combinations of *all* the contracting elevator and depressor muscles, including the masseter, temporalis, medial and lateral pterygoids commonly occur. In some cases, the torquing is so extreme that the coronoid process of the mandible gets repeatedly wrenched against the buccal aspects of the maxillary tuberosity of the patient causing unusually sited mucosal ulcers. These patients are not able to reproduce this parasomniac range of movement whilst awake!

Suggested management of REM bruxism include the following:

i. Orthotic Therapy - Mandibular Advancement Splints (Figure 2)

These are the same devices used in the management of sleep-disordered breathing to prevent upper airway collapse. They are especially effective in reducing waking symptoms in patients presenting with low frequency RMMA by preventing mandible torquing during REM SB episodes. When the upper and lower plates are tied together (Figure 2), it converts potentially injurious involuntary eccentric muscle contraction (associated with delayed onset muscle soreness), into less damaging isometric contractions.

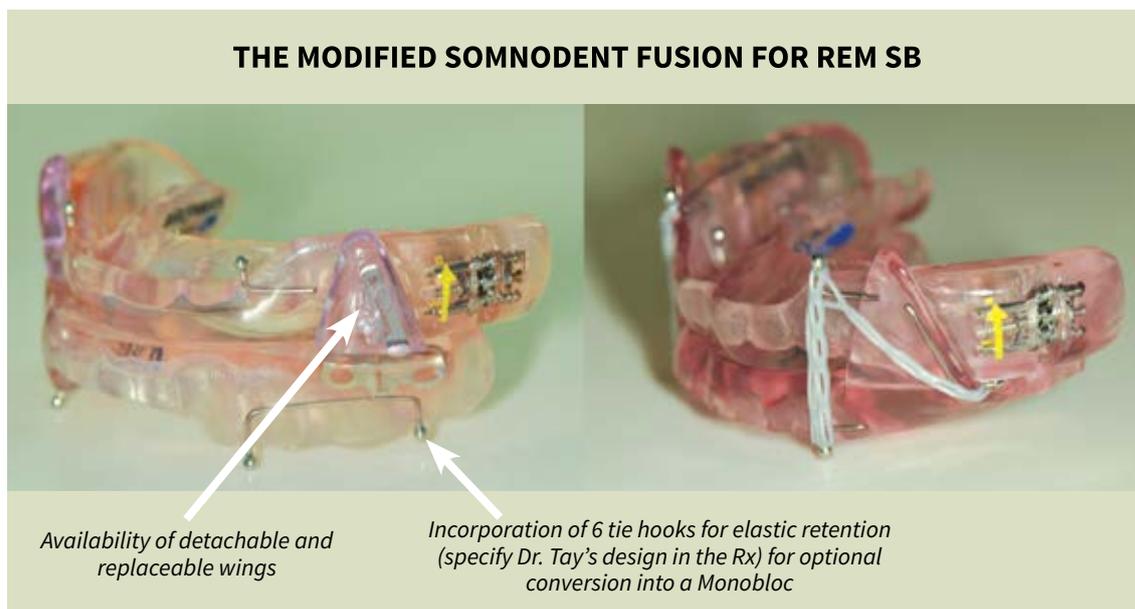
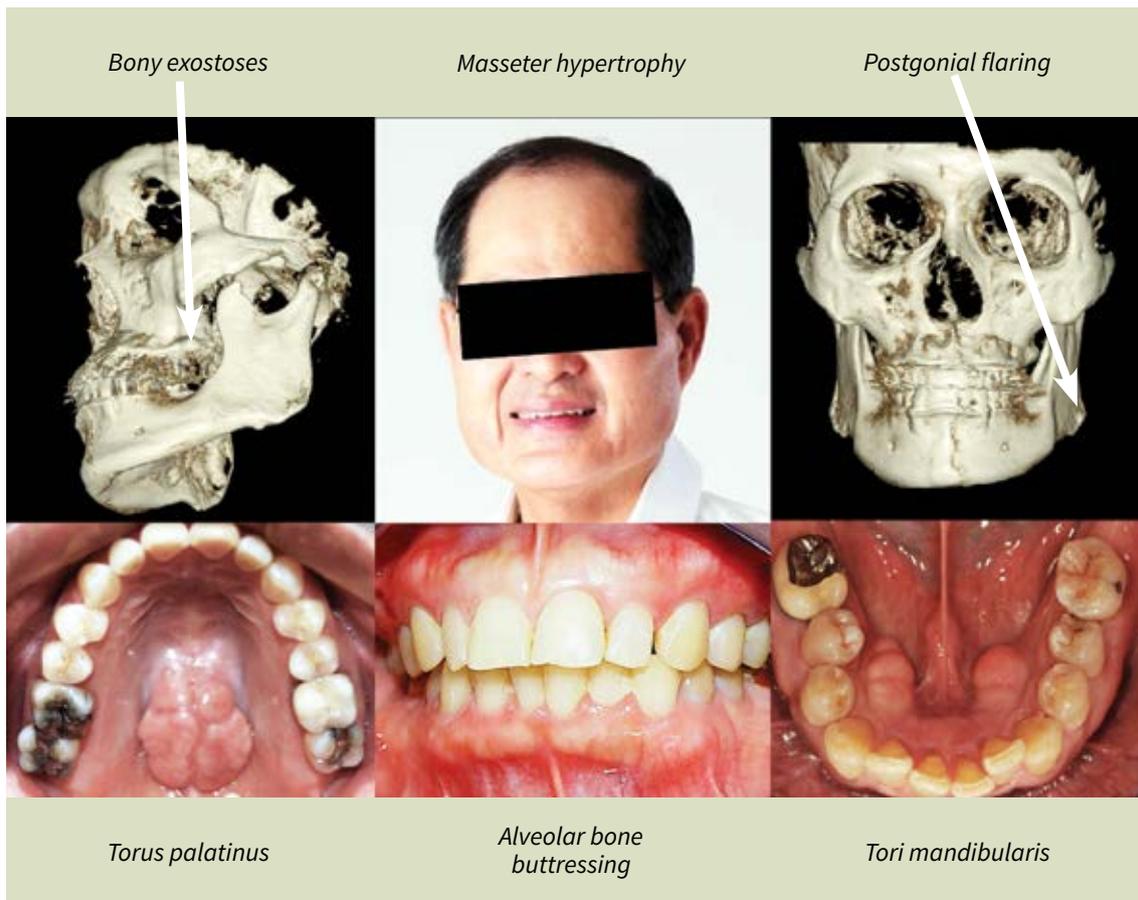


Figure 2: Orthotic therapy for REM SB

ii. Botox Injections

Although NREM-SB classically occur secondary to arousals, we have PSG documentation that some RMMA-SB episodes actually precede and cause REM arousals! It has been our experience that Botulinum toxin type A injections into the superficial masseter & temporalis muscles can stop REM arousals for up to 6 months and should be considered in patients with severe stomatognathic pain and/or associated violent and disruptive REM arousals. It should be pointed out that Botox reduces the amplitude but not the frequency of the RMMA episodes.

(C) Awake Bruxism (AB) (Figure 3)



Courtesy of Edmund Tay, Mai Hiong Endowment

Figure 3: Awake Bruxism

Approximately 1/3 of patients with SB also exhibit concomitant wake-time bruxism. This has an estimated prevalence of 12% in children and >20% in adults. Psychosocial factors like stress, anxiety-hypervigilance and personality traits have significant influence on AB. The stoic socio-cultural environment (*rewarding self-control, discouraging the open expression of emotions, keeping your mouth shut and your teeth together!*) rather than genetics are thought to be responsible for the higher prevalence of oral tori/bony exostoses in Asians.

Even though the AASM clinical diagnostic criteria for **Sleep** Bruxism (ICSD-2, 2005) listed '**Hypertrophy of the masseter muscles on voluntary forceful clenching**', it is imperative to realise that muscle hyper-

trophy in the elevator muscles can only be produced by habitual repetitive submaximal contractions occurring in association with parafunctional tooth contact when **awake** (which may exceed several diurnal hours unlike that which occurs during SB episodes). In other words, it is the wake-time parafunctional bracing/clenching that is responsible for the hypertrophied musculature, but it is during SB episodes that the resultant movement vector and/or loads generated by them are unleashed!

Despite strong clinical belief and traditional dental school teaching, awake bruxism is held by leading researchers to be the more relevant risk factor for the development of persistent myofascial TMD than sleep bruxism.

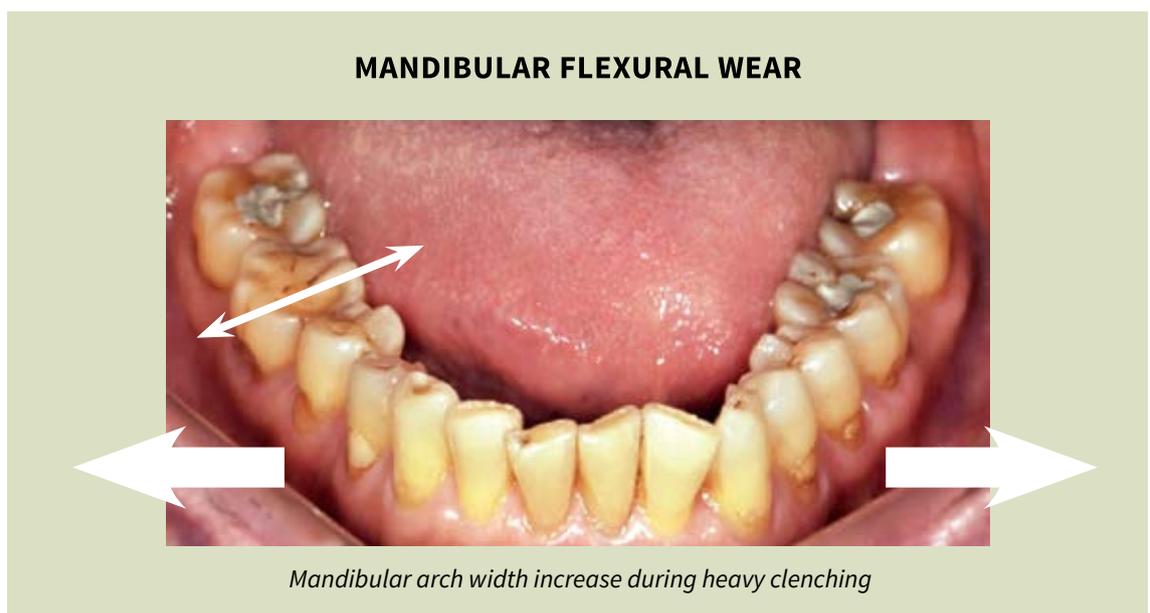
Besides masseter hypertrophy, there are several orofacial features that may be associated with AB:

i. Presence of Tori

The prevalence of **tori mandibularis** and associated jaw parafunction were reportedly higher in patients with TMD and migraine. Similarly, torus palatinus can be found in subjects who brace eccentrically and simultaneously on working and non-working (balancing) side occlusal contacts.

ii. Pathognomonic occlusal wear patterns (Figure 4)

Mandibular lateral flexure associated with parafunctional jaw clenching produces non-carious cervical abfraction lesions, dislodged fillings and pathognomonic occlusal wear patterns where the lingual cusps are cupped and higher than the buccal.



Courtesy of Edmund Toy, Mai Hiong Endowment

Figure 4: Pathognomonic occlusal wear pattern related to lateral mandibular flexure associated with habitual (awake) parafunctional clenching

iii. Buccal mucosal ridging

This is yet another interesting phenomenon associated with AB. Because the masseter muscle lies superficial to the buccinator muscle, the *linea alba* (or white line) commonly seen in chronic habitual clencher is the result of pressure necrosis of the lining buccal mucosa trapped between the horizontal fibers of the

buccinators. The masseters are engaged whenever there is *active* posterior tooth contact. Therefore, no mucosal ridging is observed wherever there is an edentulous span or break in the dental arch.

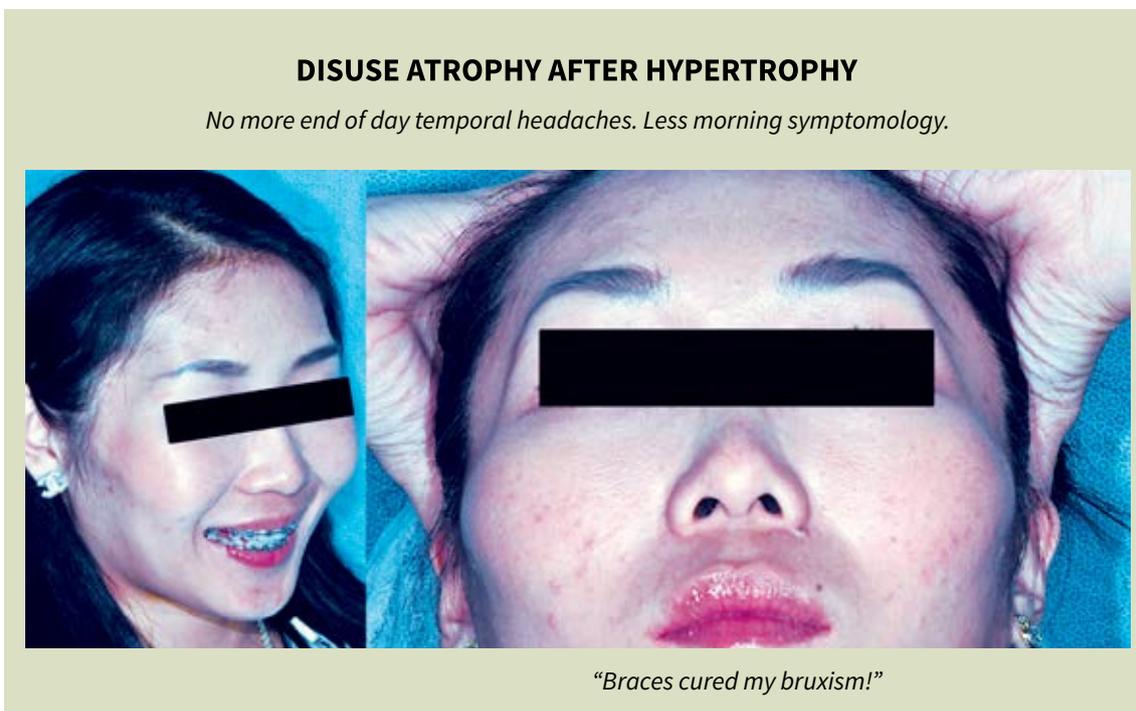
iv. Parotid-Masseter Hypertrophy Traumatic Occlusion Syndrome

The Stenson's duct which passes through the Buccinator muscles can sometimes be occluded producing a Mumps-like parotid gland swelling which characteristically lifts the earlobe. Unfamiliarity with this syndrome often results in unnecessary investigations/surgical interventions by medical colleagues.

Some possible strategies for these patients include:

- a. Teaching the patient to self-monitor this parafunctional habit. There are free mobile phone applications (e.g. *No Clenching* (Live Ideas Creative Mobile Solutions Ltd))
- b. Prescribe the use of posterior disengaging devices (e.g. NTI device) - These buccal mucosal elevations will disappear, and decrease in its severity
- c. "Chemical paralysis" (using Botox) of clenching muscles - In therapeutic Botox use, the awake bruxer is not able to continuously coactivate the muscles for 3 months or so, which results in disuse atrophy and associated weakening of the elevator muscles. Resultantly, some patients with AB would notice the improvement in frequency and severity of their daily 'end of day' jaw aches and temporal headaches.

The same phenomenon also occurs in patients with AB who are undergoing orthodontic treatment. Others may complain of the undesirable cosmetic consequences (e.g. looking gaunt) when temporalis and masseter muscles atrophy as their bracketed teeth become mobile or too uncomfortable for them to continue with their unconscious parafunctional behavior. Unfortunately, this pernicious habit resumes once the teeth regain stability after debanding. (Figure 5)



Courtesy of Edmund Toy Mai Hiong Endowment

Figure 5

(D) Secondary Bruxism

We have always to be vigilant that we're not actually dealing with a secondary bruxism i.e. comorbid sleep disorders, neurologic or psychiatric conditions, GERD, drugs/chemicals that may induce tooth grinding and/or clenching during wake or sleep.

i. Association with Upper Airway Resistance Syndrome (UARS)

UARS should always be excluded in these cases. The tell-tale signs during clinical examination and/or CBCT imaging include evidence of chronic nasal congestion (e.g. swollen nasal turbinates, a deviated nasal septum, presence of Donder's cavity), habitual mouth breathing, incorrect resting tongue posture, a restricted V-shaped maxillary arch, a deep palatal vault, retrognathia, posteriorly displaced condyles, or a forward head posture as documented on the lateral cephalogram (Tay & Pang, 2017).

ii. Association with the use of medications

Severe bruxism (both SB as well as AB) occur in children who are on methylphenidates (e.g. Ritalin, Concerta) for Attention Deficit Hyperactivity Disorder (ADHD). Despite the obvious damage to their dentition, the child continues taking the medication until they finish school or university! I have also in the past consulted with psychiatrists whose patients suffer severe phasic SB as a side-effect of serotonin specific reuptake inhibitors (SSRIs) like fluoxetine, to either alter the dosage or change to another antidepressant.

iii. Association with neurologic conditions

Because the etiology of bruxism is not fully understood, there are those who continue to advocate that the treatment of SB should be palliative. The following is one case to illustrate the error of such an approach.

I had seen a teenager with severe REM SB with complaints of severe myalgia and TMJ arthralgia on waking. She had also complained of vivid, action-filled dreams and involuntary limb movements during sleep. She did remarkably well on a stabilization splint but stopped appliance therapy after a relatively pain-free year on advice of her regular dentist who believed that her pains were psychogenic in nature. Several years later, she found me again in my private practice when all her symptoms resurged and in addition, she now presented with a huge anterior open bite. She was diagnosed with bilateral Idiopathic Condylar Resorption - at that time a relatively unknown low serum 17β -estradiol-related disorder. Together with my orthodontic colleague, we successfully managed her AOB and put her on a modified SomnoDent MAS, which worked as an orthodontic retainer whilst protecting her compromised joints from further occlusal loading during REM SB episodes. We also worked with a neurologist specializing in movement disorders, who put her on a maintenance doze of Clonazepam 1 - 1.5mg at night.

I also insisted that she undergo a full night level I PSG study. In preparation for this, she was slowly weaned off her nightly Clonazepam medication. This unmasked a late onset epileptic seizure and she was subsequently referred for a MRI scan, which discovered a dysembryoplastic neuroepithelial tumor (DNET) in left frontal lobe bordering the interhemispheric fissure. This slow growing primary brain tumour was likely responsible for epileptiform activity in the frontal lobe manifesting as REM SB-like oromotor behavior since her teenage years. Imagine how ridiculously inappropriate it would have been for us to have continued managing a brain tumor with a piece of plastic!

Conclusions

There is an urgent need for a paradigm shift in how we approach the management of the bruxing patient. One size does not fit all!

Dentists should begin to look upon RMMA-SB as a normal albeit complex, centrally regulated, oromotor behavior occurring during sleep. We have a duty of care to discover and understand the individual prevailing circumstances that may have caused this RMMA transition into its maladaptive form. Our identification of masseter hypertrophy (e.g. by increasing the amplitude of RMMA) as one such risk factor, puts the spotlight on Awake Bruxism thus making it a logical interventional target in the overall management of 'destructive' sleep bruxism and persistent myofascial TMD.

If NREM SB is a physiologic compensatory mechanism to halt airway collapse, it is essential that we place efforts to first find out the reasons for the airway challenge. Patients, including children, with NREM SB should be investigated for comorbid sleep disordered breathing, in particular UARS, if they also complain of unrestorative sleep, excessive daytime sleepiness, chronic nasal congestion, forward head posture and/or TMD. Patients exhibiting REM SB should always be closely followed up to exclude neurologic disorders like sleep epilepsy, RBD or synucleinopathies.

In the holistic and individualized management of SB, optimal results for the patient are obtained when dentists work closely with a team of dedicated sleep professionals. An overnight PSG study remains an important consideration whenever secondary bruxism is suspected. 



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The citations for the above article can be found in the 'Clinical Tips' section at www.etmh.com.sg