Sleep Bruxism and TMD - Sleep Apnea link?

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Grants: IRCS, FRQS, FCI, Chaire Recherche Canada;

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Sleep bruxism - tooth grinding:

Definition revised, Etiology, Differential diagnosis, Pathophysiology and Management avenues

Sleep Bruxism - A

Current Definition
- (Am Acad Sleep Med): Reclassified as Parasomnia
- ICSD 2 (2005): Movement Disorder
- Revisited (Lobbezoo et al, Journal of Oral Rehabilitation 2013 and ICSD 3)

Repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.

Two distinct circadian manifestations: sleep (indicated as sleep bruxism) or wakefulness (indicated as awake bruxism).
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Am Acad Sleep Med - ICSD 3 (2014): Movement Disorder

Phenotype – sub-group of SB (Rompre, J Dent Res, 2007)

- WAKE clenching in over 90% of occasionnal sleep bruxism cases
- LOW FREQUENCY of RMMA Episodes /hr of sleep: MORNING PAIN
- Low FREQUENCY of RMMA Episodes /hr in SB patients
- BELOW 4 RMMA/hr
Criteria suggested to screen patients with SB are:

1. A recent history of tooth grinding sounds occurring at least 3-5 nights per week over 6 months (if sleep alone???)

2. Presence of tooth wear (not reliable; past SB episodes)

? Morning masticatory muscle pain or headache and/or fatigue

?? Masseter muscle hypertrophy (parotid ??)

DICHOTOMY - Mismatch

Self Report questionnaire/polysomnography
Prevalence of Sleep Bruxism in a Population Sample (n=1042):
M. Maluly et al J Dent Res 2013

With questionnaires alone, the prevalence was 12.5%.

With PSG used exclusively as the criterion for diagnosis, the prevalence was 7.4% regardless of SB self-reported complaints.

The results indicated that the prevalence of SB, indicated by questionnaires and confirmed by PSG, was 5.5%.

EXPECTED since SB-Tooth Grinding fluctuate over time

124 TMD cases/46 control FEMALE subjects

**SELF REPORTS:** Tooth grinding

Told (dentist, sleep partner): 55% cases/15% Ctrl

Last 2 weeks: 15-24% cases/ 0 Ctrl

**SLEEP LAB RECORDING** (2 nights):

Positive EMG: 9.7% cases/ 10.9% Ctrl (RMMA index 1.7/1.5 hour)

2 grinding events: 60% cases/78% Ctrl

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**Prevalence**

Based on self report/ awareness from a 2\textsuperscript{nd} person

- Prevalence:
  - 8% of adult population is aware of tooth grinding sound – questionnaire studies
  - Decrease with age (child \geq 15% to elderly \leq 3% ?)
Summary of Tooth Grinding Prevalence based on Self Reports of Parents or Sleep Partner Awareness (not always precise)

Children: 14-20%

Teenagers and Adults: 12 to 8%

Over 50 years of age: 5-3%

Etiology of Primary Sleep Bruxism (SB): numerous phenotype expected (solid line: solid evidences; dash line: weaker evidences or proposed)

Autonomic and motor interactions with sleep and vigilance networks, airway & respiration?, + other

Neurotransmitters noradrenalin, dopamine, serotonin, histamine, orexin/hypocretin, acetylcholine, GABA, + other

Circadian & Ultradian Rhythm: wake/sleep, feeding cues, carry over from wake time life/anxiety, + other

Personality

Genetics: familial and environmental dominance one serotonin candidate/to confirm
Sleep Bruxism diagnosis:
EMG Recording of RMMA & DIFFERENTIAL

- EMG electrodes are placed on right and left MASSETER (belly of muscle upon voluntary clench) & right and left TEMPORALIS muscles,
- Reference on the zygomatic bone
- Minimum of one masseter or 2 masseter “jump” on one channel
- 2 EMG of R & L MASSETER are better

PLUS: Indexes of sleep leg/arm or bruxism (RMMA) movement and/or Cardiac + BREATHING (flow, O₂, apnea, etc) events with sleep stage shifts
TYPE 1: PSG & VIDEO
to discriminate different oral activities &
Rhythmic Masticatory Muscle Activity = RMMA

*Phasic and mixed type= 90 % of RMMA – not tonic/clench
- Approximately 1/3 of RMMA with tooth grinding sounds

Type 2: Portable (ambulatory)
full PSG
Compumedic, Embla (Natus), etc

Siesta
Revolutionary Diagnostics for a Wireless World

Overview
The Siesta System is a new wireless, multi-functional, ambulatory recording device. It enables recording, monitoring, storage and transfer of up to 32 physiological data inputs, such as brain, heart and muscle activity. In addition it has an Oximeter interface for heart rate and oxygen saturation as well as supporting up to 32 external DC signal inputs for recording the output of other devices such as pH meters.
Portable PSG for SB is not easy to score without video: few examples…

Carra MC – Comparison of RMMA- SB with and without video scoring.
RMMA overestimated by 23.8% without VIDEO
Also underestimated for Orofacial activities
TYPE 3: Screening- Monitoring (ambulatory)

**Few channels: breathing, EMG-RMMA/brux…**

Braebon/ MediByte, Care Fusion/Nox-T3, etc

Type IV: Sleep bruxism, one channel / Monitoring and Tx

Temporary out of usual market- SUNSTAR is now the owner

In absence of audio-video= 25 % overestimation. Carra, MC et al

Sleep and Breathing 2014

Find symptom relief from nighttime teeth grinding with GrindCare

For people who habitually grind and clench their teeth during sleep – a condition known as bruxism that affects over 5% of the population – tension headaches, jaw pain, back pain, shoulder pain, facial pain and tension are just the beginning. Many suffer from Temporomandibular Joint Disorders (TMD or TMJ) and migraines and have trouble sleeping – as do those around them. The constant friction from tooth grinding and clenching can also result in sore gums and loose teeth and destroy dental work.

GrindCare is a breakthrough solution. Unlike traditional mouthguards or nightguards and splints, it deals with the cause of teeth grinding, not just the symptoms – and is comfortable and easy to use. Using a lightweight electrode that adheres to your temple, GrindCare measures precisely how much you grind. When the device registers muscle activity, it sends a mild electrical impulse that interrupts the grinding. GrindCare does its work while you sleep without you feeling a thing – and can help break the grinding habit.

A clinical study demonstrated that GrindCare can reduce teeth grinding by 50% in just three weeks, and a user survey showed that 93% found GrindCare effective.
Example of tooth contact recorder and stimulator (BruXane, EU)

See also P McAuliffe, J Oral Rehab 2015

SB Differential Dx
Primary-idiopathic form vs. Secondary to....
SB - Hypervigilance/
Hyperarousal

(Can SB be INSOMNIA related in some patient? Yes, Maluly 2013)

WAKE carry over during sleep/
Adaptive -Maladaptive

Other directions
phenotype?

General Sleep Lab population (n=1042)
No association with DEPRESSION, OSAS, SNORING but YES with INSOMNIA (Maluly, J Dent Res 2013)

<table>
<thead>
<tr>
<th></th>
<th>No Bruxism</th>
<th>Bruxism</th>
<th>Total N</th>
<th>χ²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>% (CI 95%)</td>
<td>N</td>
<td>% (CI 95%)</td>
<td></td>
</tr>
<tr>
<td>OSAS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No OSAS</td>
<td>389</td>
<td>61.2 (65.8-93.6)</td>
<td>38</td>
<td>8.8 (6.4-14.2)</td>
<td>427</td>
</tr>
<tr>
<td>OSAS</td>
<td>180</td>
<td>91.0 (84.1-94.0)</td>
<td>18</td>
<td>9.0 (5.9-16.0)</td>
<td>198</td>
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<tr>
<td>Snoring</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No snoring</td>
<td>328</td>
<td>91.9 (89.0-95.5)</td>
<td>29</td>
<td>8.2 (4.5-11.0)</td>
<td>357</td>
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<tr>
<td>Snoring (≥x or more/ wk)</td>
<td>241</td>
<td>90.0 (81.1-92.1)</td>
<td>27</td>
<td>10.0 (7.3-18.9)</td>
<td>268</td>
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<tr>
<td>RLS</td>
<td></td>
<td></td>
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<tr>
<td>No RLS</td>
<td>441</td>
<td>90.7 (86.5-93.2)</td>
<td>42</td>
<td>9.3 (6.8-13.5)</td>
<td>483</td>
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<tr>
<td>RLS</td>
<td>101</td>
<td>88.5 (69.9-97.6)</td>
<td>13</td>
<td>11.5 (2.4-39.2)</td>
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<tr>
<td>Insomnia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No insomnia</td>
<td>508</td>
<td>92.0 (87.2-94.5)</td>
<td>44</td>
<td>8.0 (5.5-12.8)</td>
<td>552</td>
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<tr>
<td>Insomnia</td>
<td>61</td>
<td>83.5 (93.6-82.9)</td>
<td>12</td>
<td>16.5 (6.4-17.1)</td>
<td>73</td>
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<tr>
<td>Anxiety</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No anxiety</td>
<td>482</td>
<td>92.6 (90.1-95.1)</td>
<td>38</td>
<td>7.4 (4.9-9.9)</td>
<td>520</td>
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<tr>
<td>Anxiety</td>
<td>37</td>
<td>88.0 (69.7-94.3)</td>
<td>5</td>
<td>12.0 (5.7-30.3)</td>
<td>42</td>
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<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No depression</td>
<td>468</td>
<td>92.6 (90.0-94.7)</td>
<td>37</td>
<td>7.4 (5.3-10.0)</td>
<td>505</td>
</tr>
<tr>
<td>Depression</td>
<td>47</td>
<td>88.9 (78.5-96.2)</td>
<td>6</td>
<td>11.4 (3.8-21.5)</td>
<td>53</td>
</tr>
</tbody>
</table>

OSAS = Obstructive Sleep Apnea Syndrome; RLS = Restless Legs Syndrome.
Learned activity/
Brain plasticity/
adaptive state

Patients with SB = larger cortical activation (MEG mapping) when they execute a VOLUNTARY clenching or chewing motor task during WAKE time

Kervancioglu BB et al J Sleep Res 2012

Psychophysiological aspects: debated
Role of life pressure ???

• In a large population (n = 100), psychosocial stress during wakefulness does not seem to influence masticatory EMG during sleep (Pierce 1995): Role of anxiety (coping style), personality ?

• In a cross-sectional telephone survey, patients reporting tooth grinding during sleep were found “DMS-IV anxiety disorder” with a low odds ratio:1.3 (Ohayon 2001)
SB with Pathology is a different story:

**Secondary Bruxism**: concomitant to disorder or disease, after brain trauma or medication

**Relief of Wake Bruxism with D2 and 5Ht 3 antagonist medication: Metoclopramide**

**Wake time BRUXISM AFTER BRAIN TRAUMA**
(hemorrhage in frontal lobe) / n of 2, no EMG
H.S. Yi et al, Ann Rehab Med 2013

**Differential Dx critical - **SECONDARY SB

Concomitant Neurological sleep disorders:

- **Oromandibular myoclonus/tooth tapping in 10% of Sleep bruxism subjects (Kato T, 1999):**

- **REM behavior disorder** (Sleep bruxism and mainly Oromandibular Myoclonus found in RBD subjects; Abe, Sleep Med 2013)

- **Epilepsy** (Vetrugno R 2002), Parkinson’s, Huntington’s, Oromandibular dystonia or Neuroleptic induced dyskinesia……
Tooth Tapping
= Sleep oromandibular or orofacial myoclonus (found in 10% of SB patients)
Important to EXCLUDE SLEEP EPILEPSIA

AGAIN SB and Concomitant Neurological-Movement sleep disorders:

Periodic Limb Movement Syndrome
Mostly leg, about 40% in arm (+ 10 mvt/hr)
- RLS (the wake variant of PLMS) and SB= 10% overlap (pop survey, Lavigne & Montplaisir 1994)
- 37% of RLS patients have bruxism (Wake or Sleep/ quest only?) with concomitant migraine (84% of brux cases?) & improve with dopaminergic medication (pramipexole, ropimirole) (clinical pop, Dickoff D -Abst- NEUROLOGY meeting 2015)
- A 3 sec temporal association of SB, PLM and Arousal was found suggesting some commonality in mechanism (van der Zaag 2014)
SB - Interaction or CAUSALITY between orofacial pain and TMD / SLEEP bruxism and/or breathing?

Temporomandibular Disorder and Morning Transient OroFacial Pain

More Sleep Bruxism EMG activity does not = More PAIN
No more RMMA contraction in Morning Transient Pain (Abe S, JOFP, 2013)

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Sleep and RMMA Parameters for Controls (CTRL), Sleep Bruxers with Pain (SBrP), and Sleep Bruxers without Pain (SBrN)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>CTRL</strong> (n = 19)</td>
</tr>
<tr>
<td>Age</td>
<td>24.05 ± 1.26</td>
</tr>
<tr>
<td>Sex</td>
<td>8 F/11 M</td>
</tr>
<tr>
<td>Sleep stage shift</td>
<td>241.32 ± 15.35</td>
</tr>
<tr>
<td>Microarousals/hr</td>
<td>9.33 ± 2.02</td>
</tr>
<tr>
<td>RMMA episodes</td>
<td></td>
</tr>
<tr>
<td>Episodes/hr</td>
<td>1.34 ± 0.22</td>
</tr>
<tr>
<td>Phasic episodes/hr</td>
<td>1.00 ± 0.23</td>
</tr>
<tr>
<td>Tonic episodes/hr</td>
<td>0.03 ± 0.02</td>
</tr>
<tr>
<td>Mixed episodes/hr</td>
<td>0.31 ± 0.09</td>
</tr>
<tr>
<td>Episodes with noise</td>
<td>0.37 ± 0.17</td>
</tr>
</tbody>
</table>

*ANOVA; Tukey test*
Comparison of the EMG data (# of EMG events per hour of sleep) between different groups – ONE CHANNEL EMG: temporalis

Yachida W et al. J DENT RES 2012;91:562-567

Sustained activity / periodic transient one
Wake / sleep time carry over influences

In TMD cases= pain due to… ??

Elevated - Sustained Activity in all sleep period for 72% of TMD cases (n:124/ 42 Ctl)
(K Raphael, JOR 2013)

Background EMG during non-SB event periods is significantly higher for women with myofascial TMD (median = 331 uV and mean = 498 uV) than for control women (median = 283 uV and mean = 388 uV)

Background EMG was positively associated associated with pain intensity AWAKE – CARRY OVER ?
WHILE RMMA-SB event related EMG was negatively…
Sustained activity / periodic transient one
Wake / sleep time carry over influences

Sleep bruxism = no relation of
**TRANSIENT Rhythmic** Masticatory Muscle
activity and pain (previous slides...)

Some **WAKE carry over influence persist**?
Trait Anxiety is associated to longer duration of
masseter & temporalis Muscle Activity for 1<sup>st</sup> hr
of sleep; also correlated to temporalis for all
sleep duration (n=15 non pain subjects; Manfredini JOR 2011)

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Differential Dx critical -
Concomitant sleep disorders breathing:

**QUESTIONNAIRE only:**
*IF TMD= 4% S&S of OSA with OR= 3.6 for
chronicity of TMD pain* (Sanders, JDR, 2013 – OPPERA study)

**Table 4.**
Multivariable Model Showing Odds Ratios (95% confidence limits) for Chronic TMD, OPPERA Case Control Study (n = 1,716), 2006-2008

<table>
<thead>
<tr>
<th></th>
<th>Model 1 OR (95%CL)</th>
<th>Model 2 OR (95%CL)</th>
<th>Model 3 OR (95%CL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High likelihood of obstructive sleep apnea</td>
<td>3.48 (1.95, 6.19)</td>
<td>3.34 (1.87, 5.96)</td>
<td>3.63 (2.03, 6.52)</td>
</tr>
</tbody>
</table>
Differential Dx critical - Concomitant sleep disorders breathing:

QUESTIONNAIRE and Sleep Recording:

See next slides

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TMD population: RERA are higher in TMD female than in Control Subjects (B Dubrosky, J Clin Sleep Med 2014)

<table>
<thead>
<tr>
<th>PSG Parameters</th>
<th>TMD Patients (n=124)</th>
<th>Controls (n=46)</th>
<th>p value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Continuity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Sleep Time (TST, min)</td>
<td>386.4 ± 53.1</td>
<td>402.3 ± 45.8</td>
<td>0.139</td>
</tr>
<tr>
<td>Sleep Efficiency (SE) = TST/TTRx100%</td>
<td>89.7 ± 8.7</td>
<td>92.3 ± 6.4</td>
<td>0.128</td>
</tr>
<tr>
<td>Sleep Onset Latency (SOL) (min)</td>
<td>11.6 ± 16.5</td>
<td>9.6 ± 10.1</td>
<td>0.276</td>
</tr>
<tr>
<td>Number of Awakenings</td>
<td>18.2 ± 11.3</td>
<td>14.3 ± 7.8</td>
<td>0.069</td>
</tr>
<tr>
<td>Sleep Architecture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1 as % of TST</td>
<td>12.2 ± 7.6</td>
<td>9.2 ± 5.0</td>
<td>0.034</td>
</tr>
<tr>
<td>N2 as % of TST</td>
<td>51.9 ± 10.6</td>
<td>51.1 ± 10.3</td>
<td>0.898</td>
</tr>
<tr>
<td>N3 as % of TST</td>
<td>16.5 ± 10.9</td>
<td>19.0 ± 8.5</td>
<td>0.356</td>
</tr>
<tr>
<td>REM as % of TST</td>
<td>19.3 ± 6.9</td>
<td>20.6 ± 5.7</td>
<td>0.324</td>
</tr>
<tr>
<td>REM Latency (min)</td>
<td>109.1 ± 64.3</td>
<td>88.6 ± 52.1</td>
<td>0.047</td>
</tr>
<tr>
<td>Arousals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous Arousal Index</td>
<td>10.6 ± 6.0</td>
<td>12.0 ± 5.6</td>
<td>0.195</td>
</tr>
<tr>
<td>Respiratory Arousal Index</td>
<td>6.0 ± 6.1</td>
<td>3.5 ± 3.3</td>
<td>0.021</td>
</tr>
<tr>
<td>PLM Arousal Index</td>
<td>1.1 ± 2.4</td>
<td>1.6 ± 3.3</td>
<td>0.295</td>
</tr>
<tr>
<td>Total Arousal Index</td>
<td>17.8 ± 8.2</td>
<td>17.0 ± 6.8</td>
<td>0.833</td>
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<tr>
<td>Respiratory variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apnea-hypopnea index (AHI)</td>
<td>3.7 ± 6.6</td>
<td>2.4 ± 3.9</td>
<td>0.504</td>
</tr>
<tr>
<td>Respiratory disturbance index (RDI)</td>
<td>8.1 ± 8.5</td>
<td>5.6 ± 5.1</td>
<td>0.016</td>
</tr>
<tr>
<td>RERA index total</td>
<td>4.3 ± 4.3</td>
<td>2.6 ± 2.7</td>
<td>0.017</td>
</tr>
<tr>
<td>PLM Index</td>
<td>4.9 ± 10.3</td>
<td>5.8 ± 13.8</td>
<td>0.424</td>
</tr>
</tbody>
</table>

*p between-group comparisons from ANOVA that included BMI and age as covariates.
Sleep bruxism

Excessive RMMA and clenching during sleep?

Headache

Comorbid sleep disorders?

Re-opening upper airways?

Lubrication of oropharynx?

Arousal reaction?

Sleep-disordered breathing

Hypoxia and hypercapnia?

Sleep fragmentation?

Fig. 3. Comorbid SB, headache, and SDB: putative mechanisms.


Table 2—Rates of International Classification of Sleep Disorders, Second Edition (ICSD-2) Diagnoses of Temporomandibular Joint Disorder (N=53)

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>Male (n=10)</th>
<th>Female (n=43)</th>
<th>Total (N=53)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
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<tr>
<td>Insomnia</td>
<td>2</td>
<td>20</td>
<td>9</td>
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<tr>
<td>Idiopathic</td>
<td>0</td>
<td>-</td>
<td>3</td>
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<tr>
<td>Primary Insomnia (DSM-IV-TR)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>(Includes psychophysiological)</td>
<td>2</td>
<td>20</td>
<td>12</td>
</tr>
<tr>
<td>Insomnia due to TMD</td>
<td>0</td>
<td>-</td>
<td>3</td>
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<tr>
<td>Insomnia due to Mood Disorder</td>
<td>0</td>
<td>-</td>
<td>2</td>
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<tr>
<td>Any Insomnia Diagnosis</td>
<td>2</td>
<td>20</td>
<td>17</td>
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<tr>
<td>Obstructive Sleep Apnea</td>
<td>2</td>
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<td>9</td>
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<tr>
<td>Mild (RDI = 5-14.9)</td>
<td>2</td>
<td>20</td>
<td>0</td>
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<tr>
<td>Moderate (RDI = 15-29.9)</td>
<td>1</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>Severe (RDI ≥ 30)</td>
<td>1</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

Sleep Lab, TMD population

- **35.8% INSOMNIA**
- **28.4% OSA**
- **17.3% SLEEP BRUXISM**

Smith, Sleep 2009

- **45%** of TMD patients 1 sleep disorder
- **26%** of TMD patients 2 sleep disorder
- **17%** of TMD patients 3 sleep disorder
SB - Brainstem and/or Cortical Generator of SB-RMMA Activity during SLEEP

Pour la Science, août 2004
Huynh et Lavigne

Wake voluntary mastication

Sleep Bruxism: onset within Cortical arousal but generated in brainstem
Sleep arousal (natural mechanism)

Transition periods (3-15 seconds) with rise in brain, heart and respiratory activities plus in muscle tone

Can be a preparatory flight or fight activation of primitive brain during sleep = protective role for survival!

SB & ANS: 1st evidences from Satoh & Harada 1973

NB: In Europe: microarousal;
In North America-USA: arousal

SEQUENCE of physiologic events before tooth contact

Sequence of Physiological Activities Associated to RMMA-SB Episodes in PRESENCE of Sleep Arousal (observed with 50-80% of Episodes)

- 4 to 8 min:
  Rise in Autonomic Cardiac Sympathetic Dominance

- 4 sec:
  Rise in Brain CX Activity (EEG)

- 1 sec:
  Tachycardia
  Big Breath
  Rise in Supra Hyoid Muscle Tone

Swallowing
(less than half of episode)

ONSET OF RMMA with possible TOOTH GRINDING with Rise in Blood pressure

RMMA Episode

Cascade of autonomic activation:
Rise in blood pressure (20%) with sleep bruxism events (A Nashed, SLEEP 2012)

Risk if already Hx high blood pressure is UNKNOWN

Awarded by SLEEP – APSS meeting 2013
The Challenge – proof of concept

Medication that may reduce Autonomic Cardiac Activity: 
see next slide

Medication that may reduce Autonomic Cardiac Activity: 
see next slide

Management Pharmacologic Approaches

Cardioactive (proposed by Sjoholm):
1- Propranolol NO EFFECT in Experimental RCT

BUT
2- Clonidine 0.3 mg:
60% reduction but hypotension in 20% of subjects (Huynh et al, SLEEP 2006)

Unpublished data coming from Baba, Japan with 0.1 mg
SB- FOCUS on airway & respiration; May be related in some patient? Again, it is not explaining all causes of SB.

RMMA and BREATHING: Flow and Oxymetry (O₂)
Airway in sleep: tongue and mandible tend to move downward and backward

Oropharyngeal domain...

Cistulli et al Sydney, Australia

Sleep disorders breathing crescendo

- Occasional snoring
- Habitual snoring
TO
- Upper airway resistance syndrome-
- \( \text{RERA} = \text{Respiratory Event Related Arousal} \)
TO
- Occasional apneas or hypopneas
- Obstructive Sleep Apnea (OSA) syndrome

RISK OF:
Metabolic syndrome (diabetes, hypertension, obesity)
TO
Car accidents, cardiovascular problems, etc
Intersecting prevalence with age may explain why you see association in your practice:
Sleep Bruxism decrease
Sleep Apnea increase

AHI 15 and over:
↑ 9.5% to 17.4%
Peppard 2013

SB ↓ 12% to 3%
Lavigne & Montplaisir
Sleep 1994

M. Maluly et al, J Dent Res 2013
Sleep lab (1 night) =
AHI same & SaO2 no difference
IN SB PATIENTS
(large population and large age range/cluster – sub goup)
Temporal association

Cause and effect: *Cause should precede the effect*
- RMMA and Apnea or hyponea timing
- Can be experimentally reproduced
  Or
*Altered by treatment*

What is first SB-RMMA or Apnea? Miku Saito et al, Hokkaido University, Sapporo, Japan (J Sleep Res 2014)

Flow
Effort (Tho)
Effort (Abd)
SpO2
Mass

Apnea  55% to Sleep Bruxism

T1 (AHE to SBE)  10s
The Challenge – proof of concept

Opening airway reduce RMMA
see next slides

NOT ALL…

20% of SBE not associated to Apnea events
Lower # RMMA episodes/hr with occlusal (bite) splint (lower) and a MAA (Silencer, BC)
(A. Landry-Schonbeck, Int J Prosthodont 2009)

Mild benefit
For SB patients
No fracture of MAA
SHORT TERM

Lower RMMA index
57%

Less Headache in the morning
MC CARRA – SLEEP MEDICINE 2013
OFF LABEL / NOT FDA – PRE ORTHODONTIC PATIENTS

BSL= baseline night
A= free splints
B= MAA in central occlusion
C= MAA advanced (50%)

Morning headache
VAS
0 5 10 15 20 25 30 35

0.04
0.02
0.07
0.4
RMMA episode index
0 1 2 3 4 5

For SB patients
No fracture of MAA
Clinical association is not causality

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>Male (n=10)</th>
<th>Female (n=43)</th>
<th>Total (N=53)</th>
</tr>
</thead>
<tbody>
<tr>
<td>INSOMNIA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychophysiological</td>
<td>2 20%</td>
<td>9 20.9%</td>
<td>11 20.8%</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>0 -</td>
<td>3 7%</td>
<td>3 5.7%</td>
</tr>
<tr>
<td>Primary Insomnia (DSM-IV-TR)</td>
<td>2 20%</td>
<td>12 27.9%</td>
<td>14 26.4%</td>
</tr>
<tr>
<td>Insomnia due to TMD</td>
<td>0 -</td>
<td>3 7%</td>
<td>3 5.7%</td>
</tr>
<tr>
<td>Any Insomnia Diagnosis</td>
<td>2 20%</td>
<td>17 39.5%</td>
<td>19 36.8%</td>
</tr>
<tr>
<td>OBSTRUCTIVE SLEEP-APNEA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild (RDI = 5-14.9)</td>
<td>2 20%</td>
<td>9 20.9%</td>
<td>11 20.8%</td>
</tr>
<tr>
<td>Moderate (RDI = 15-29.9)</td>
<td>2 20%</td>
<td>3 7%</td>
<td>7 13.2%</td>
</tr>
<tr>
<td>Severe (RDI ≥ 30)</td>
<td>1 10%</td>
<td>1 2.3%</td>
<td>2 3.8%</td>
</tr>
<tr>
<td>Any Sleep Apneas DX</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MANDIBULAR DISORDERS</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Sleep Lab, TMD population & COMORBIDITIES** SMITH, SLEEP 2009

- **35.8% INSOMNIA**
- **26.4% OSA**
- **17.3% SLEEP BRUXISM**
  - 45% of TMD patients 1 sleep disorder
  - 26% of TMD patients 2 sleep disorder
  - 17% of TMD patients 3 sleep disorder

SB- Genetic… no gene therapy for bruxism
Phenotyping SB patients and blood relatives for genetics polymorphism

**QUESTIONNAIRE STUDY:**

- 49% of male and 64% of female SB phenotype variance is due to Genetic and environmental factors: Hublin et al 1998 J Sleep Res (2419 heterozygotic twins; 1298 homozygotic twins))

- Genetic factors account for half of the phenotypic variance in liability to sleep-related bruxism in young adults: a nationwide Finnish twin cohort study. 
  Rintakoski K et al 2012

**SLEEP LAB STUDY:**

37% of mild and severe SB subjects (EMG frequency) have one direct blood relative with tooth grinding Hx = suggest modest hereditary effect (Khoury et al, submitted; Montreal SB population (n=111 with 2 nights of sleep)

Ambulatory one channel EMG study – limited discrimination: An association of serotonin receptor (C allele carrier HTR2A) and bruxism RR=4.2 (Abe Y from Baba labs; J Sleep Res 2012)

Not a single gene expected: See the OPERRA TMD Study: 202 phenotypes and 5 gene candidates; Smith, J Pain 2013
VARIous management for SB: *Effect & Level of evidences*
*Winocur, in Sleep Med for Dentist, Quintessence, 2009*

**Behavioral management approaches:**
- Explanation of causes and exacerbation factors for SB
- Elimination of clenching teeth and bracing jaw during daytime in reaction to life pressures
- Lifestyle changes; introduction of sleep hygiene, relaxation, autohypnosis, and winding down before sleep
- Physical therapy and training in relaxation and breathing
- Psychologic therapy to manage stress and life pressure

**Questionable effect – Weak evidence so far but patient report subjective well being!**
Management with oral appliances

Splint studies = CRITICAL PERIOD to monitor changes in EMG level over time
(Muscle fibers length = adaptation / Motoneurons activity “stabilisation”?)

van der Zaag, JOP 2005
1st night= 6.2-7.4
4 week later= 11.1- 10.6
Orofacial EMG index/hr
No video

Harada et al.
J Oral Rehab 2006
6 weeks
“Cyclic variation”
Orofacial EMG estimation
ambulatory / no video

Dubé, JDR, 2004
2 weeks ONLY!
RMMA index & Laboratory

Baseline  W 1  W 2  W 4
0 1 2 3 4 5 6 7 8 9
Orofacial EMG index/hr

1st night
Breathing / AHI aggravation in adults with Occlusal Splint
(Gagnon et al, Int J Prostho 2004)

Findings reproduced
Nikolopoulou M et al
JOFP, 2013

Study 2: # RMMA episodes/hr with occlusal (bite) splint (lower) and a MAA (Silencer, BC)
(A. Landry-Schonbeck, Int J Prostho 2009)

No fracture of MAA in these severe SB patients
SHORT TERM
Frequent headache (as a complaint)

**Headache in the morning**

- Frequent headache in the morning: 57% of cases

**Effect & Level of evidences** – Winocur, *Sleep Med for Dentist, Quintessence, 2009*

**Sedative and muscle relaxants:**
- Clonazepam: Positive effect - Moderate evidences - Risk of dependence
- Diazepam, buspirone: Positive effect based on Case reports - Risk of dependence

**Serotonin-related:**
- Tryptophan: No effect
- Amitriptyline: No effect in RCT

**Dopaminergic:**
- Levodopa: Modest effect in RCET (30%) – Moderate evidences
- Pergolide: Positive effect - Case report – implant related!
- Bromocriptine: No effect in RCET

**Cardioactive:**
- Clonidine: Positive effect in RCET – Moderate evidences - risk of hypotension in morning – MEDICAL supervision and lowest dose
- Propranolol: No effect in RCET
Botulinum Toxin reduces the intensity rather than the generation of the contraction in jaw-closing muscles.

*Amplitude is smaller, not less SB-RMMA*

*So the generator remain active SUGGESTING a Central Origin*

Vibration or as below, electirc schock, reduce RMMa-SB (Jadihi F, J Oral Rehab 2008)

Exterosupressive Suppression
Example of tooth contact recorder and stimulator (BruXane, EU)

See also P McAuliffe, J Oral Rehab 2015

Role of OCCLUSION?
Manfredini M J Oro Facial Pain 2012
Lateroretrusive + p=0.03
But only 4.6% Variance of BS & Occlusion
Low PREDICTIVE value for Tx

After controlling for 16 variables
Of occlusion= NO relation with SB
Ommerborn M; Int J Oral Sc 2012
Positional therapy reduce apnea-hypopnea? For sleep bruxism Heinzer, Lavigne et al, Sleep Med 2012

SUMMARY of Management in Presence or Absence of Sleep Disorder Breathing (SDB)

**Clinical INDICATORS:**
- Tooth Grinding Sounds (current?)
- Awareness of Clenching
- Tooth Wear (not reliable for current SB)

Sleep Recording of at least one Masseter muscle revealing:
- Mild frequency of SB (2-4 RMMA episode/hr)
Or
- Moderate to high frequency of RMMA (4 or + RMMA episode/hr)

**Presence of Sleep Disordered Breathing**

**Absence of Sleep Disordered Breathing**
- Cognitive Behavioral Treatment (modest level of evidence)
- Occlusal Splint (no if SDB)
- Medication: clonazepam, clonidine, botulinum toxin (short term, low dose & medical supervision)

**Sleep Position Trainer**

ENT and/or Orthodontic Examination and Treatment (when needed)

Mandibular Advancement Appliance Or CPAP With or without medication (see below)

Yellow or Green Light