Sleep Bruxism and TMD- Sleep Apnea link?

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Disclosure:

Relationships without and with commercial interests:

- Grants/Research Support: CRC, CIHR, FRQS
- Lecture in Europe for ResMed
- President of Canadian Pain Society
- Free oral appliances for research use (no control on data): ResMed, Somnomed and Recording tools: Braebon

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Sleep bruxism - tooth grinding:

Sleep Bruxism - A

Current Definition

(Am Acad Sleep Med):

- **ICSD 1: Parasomnia**
- **ICSD 2 (2005): Movement Disorder**

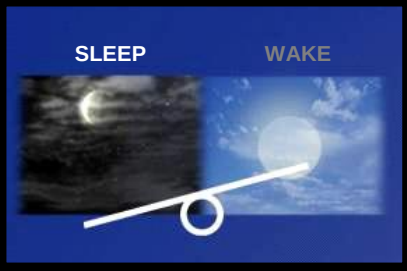
- **Revisited (Lobbezoo et al, Journal of Oral Rehabilitation 2013 and ICSD 3)**

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).

2 type of bruxism OR continuum of same

Current Definition (Lobbezoo et al, 2013 and ICSD 3)

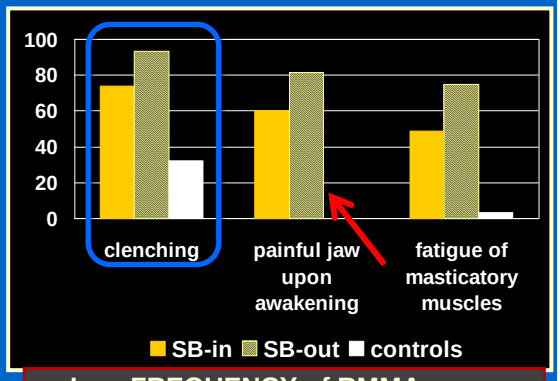


Two distinct circadian manifestations:
sleep (indicated as sleep bruxism)
or wakefulness (indicated as awake bruxism).

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**

Sleep bruxism and TMD: Weak Relationship - dichotomy of self report / sleep lab (Raphael, et al, JADA, 2012)

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):

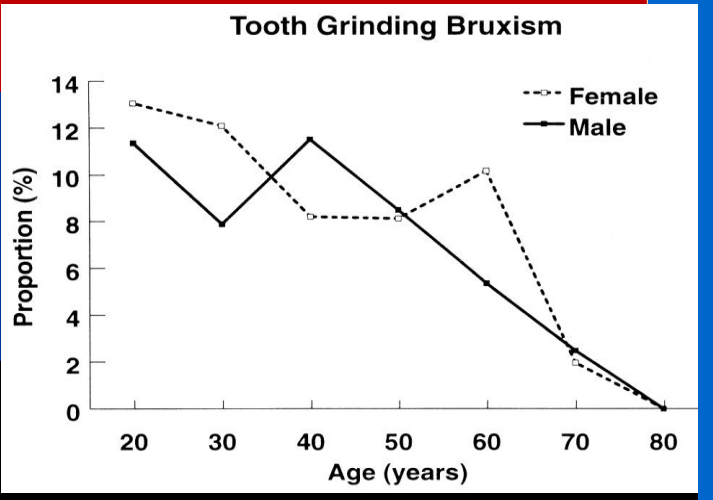
Positive EMG: 9.7% cases/ 10.9% Ctrl (RMMA index 1.7/1.5 hour)

2 grinding events: 60% cases/78% Ctrl

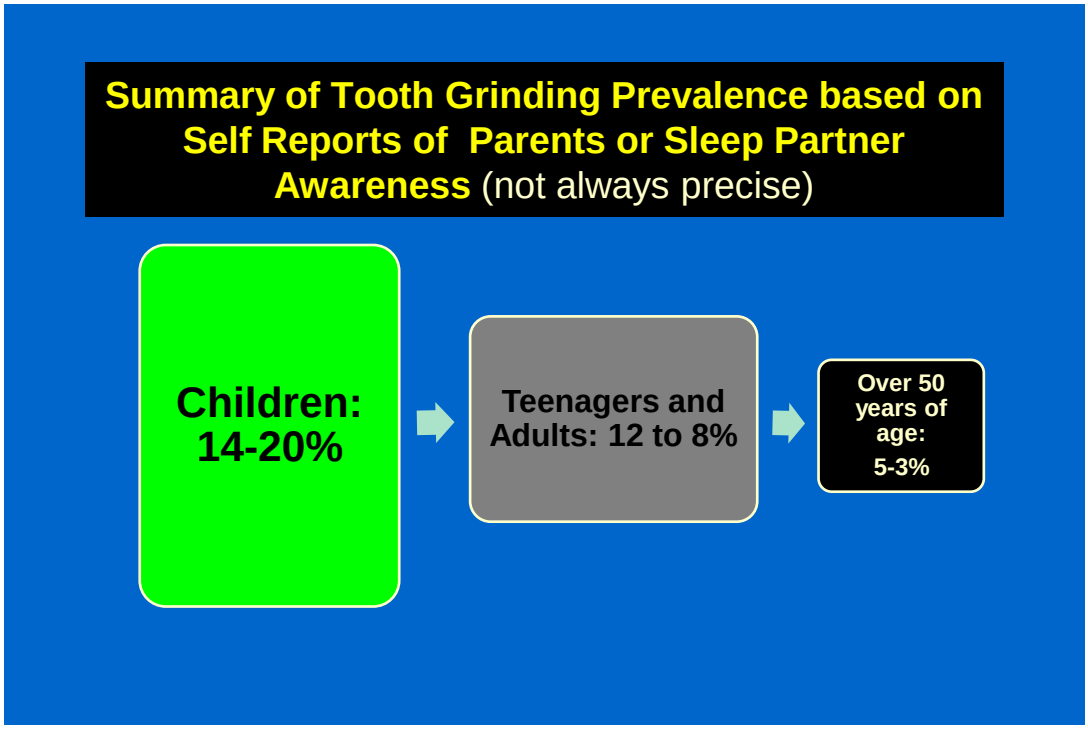
Prevalence

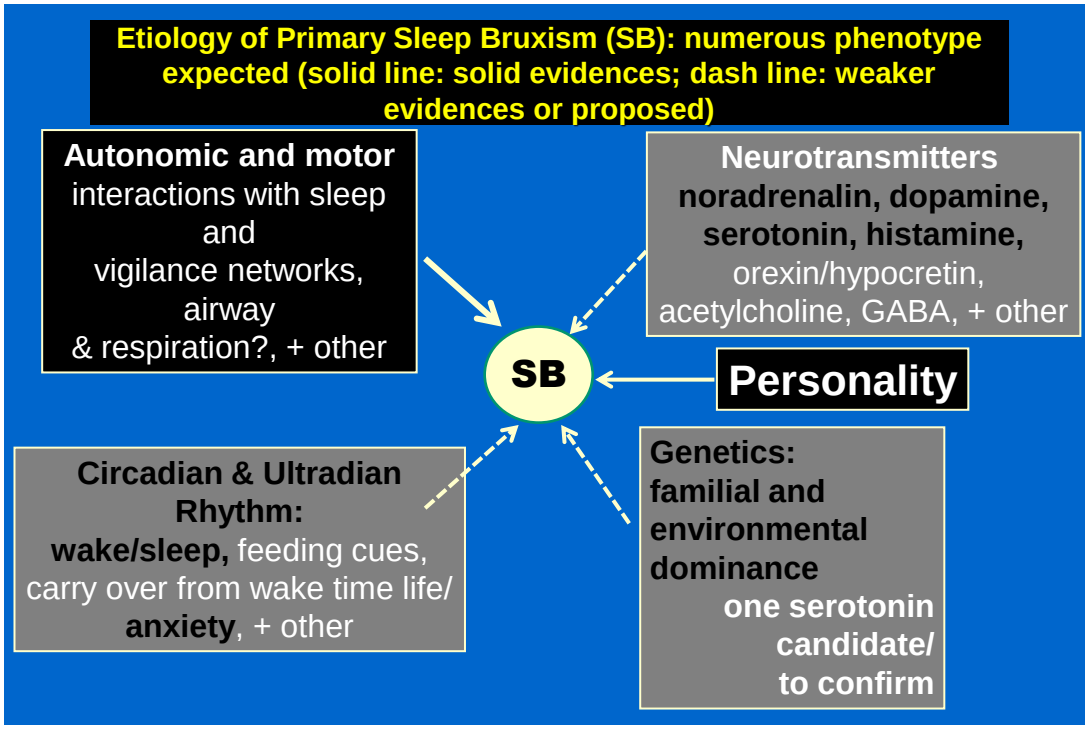
Lavigne 1992, Carra 2011

Based on self report/ awareness from a 2nd person



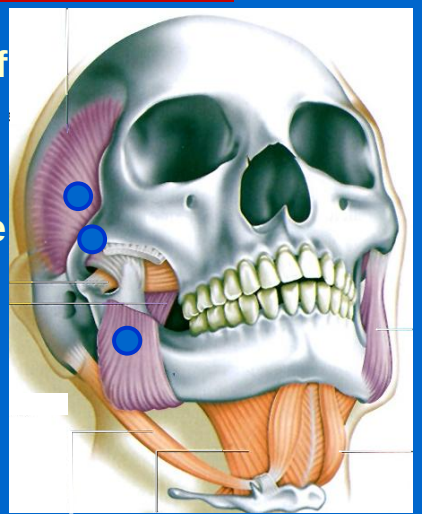
- **Prevalence:**
  - **8% of adult population is aware of tooth grinding sound – questionnaire studies**
  - **Decrease with age (child ≅ 15% to elderly ≅ 3% ?)**


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%

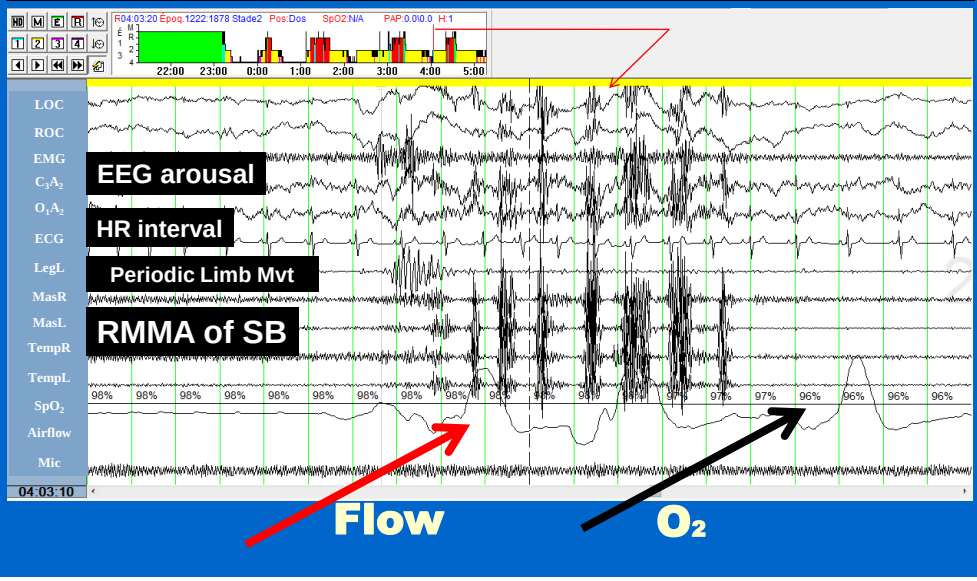

Etiology of Primary Sleep Bruxism (SB): numerous phenotype expected (solid line: solid evidences; dash line: weaker evidences or proposed)
Autonomic and motor interactions with sleep and vigilance networks, airway & respiration?, + other
Neurotransmitters noradrenalin, dopamine, serotonin, histamine, orexin/hypocretin, acetylcholine, GABA, + other
SB
Personality
Circadian & Ultradian Rhythm: wake/sleep, feeding cues, carry over from wake time life/ anxiety, + other
Genetics: familial and environmental dominance one serotonin candidate/ to confirm

Sleep Bruxism diagnosis: EMG Recording of RMMA & DIFFERENTIAL

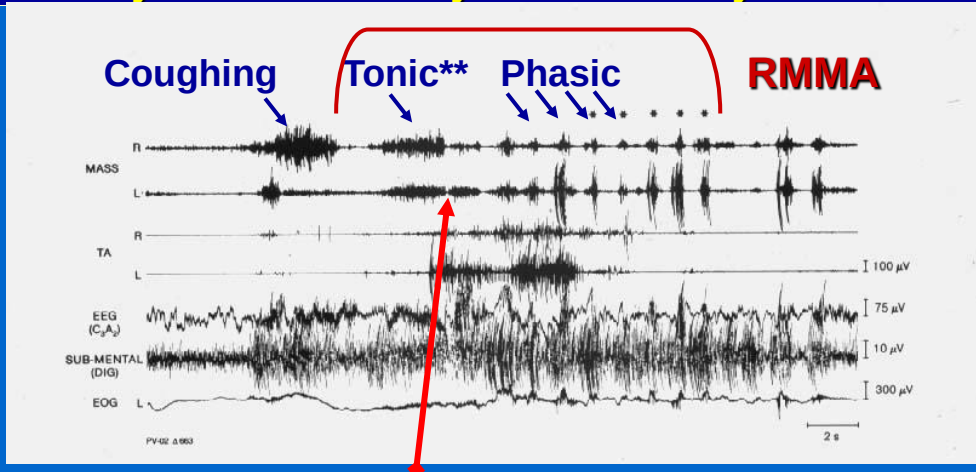
- EMG electrodes are placed on right and left MASSETER (belly of muscle upon voluntary clench) & right and left TEMPORALIS muscles,
- Reference on the zygomatic bone
- Minimum of one masseter or 2 masseter "jump" on one channel
- 2 EMG of R & L MASSETER are better



PLUS : Indexes of sleep leg/arm or bruxism (RMMA) movement and/or Cardiac + BREATHING (flow, O_2, apnea, etc) events with sleep stage shifts



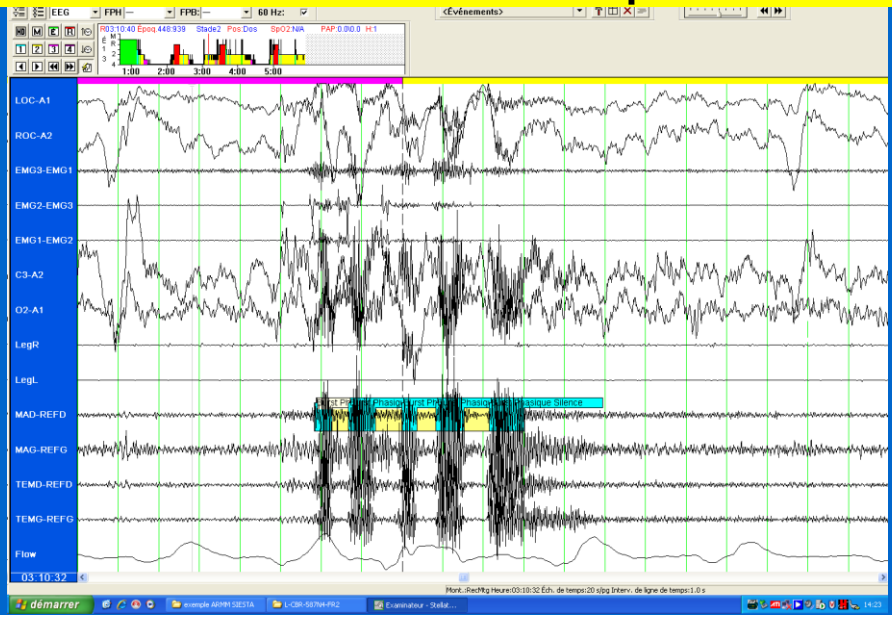
TYPE 1: PSG & VIDEO
to discriminate different oral activities &
Rhythmic Masticatory Muscle Activity = RMMA



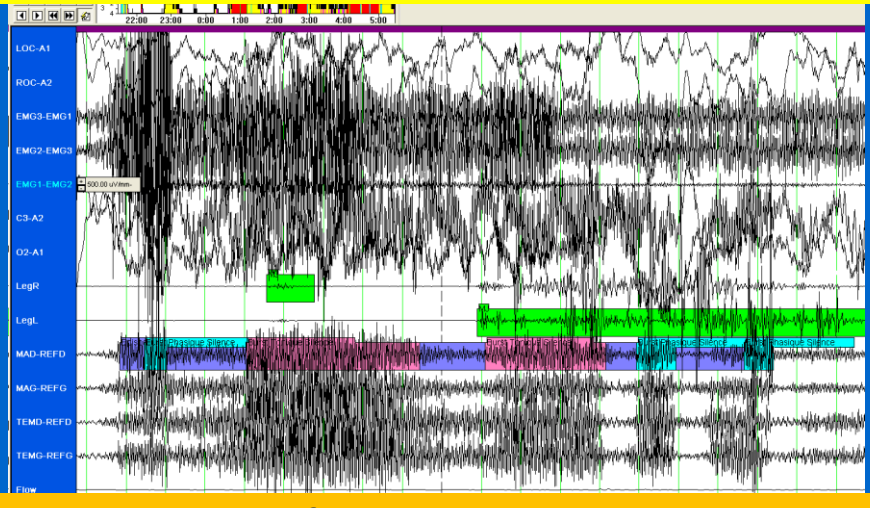
Phasic and mixed type= 90 % of RMMA – not tonic/clench
- Approximately 1/3 of RMMA with tooth grinding sounds

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

Portable PSG for SB is not easy to score without video: few examples…



Portable PSG not easy to score without video



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/ Medi*Byte*, Care Fusion/Nox-T3, etc

The NOX-T3 portable sleep monitor maximizes patient comfort and clinical value.

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

GrindCare Because better days start with good nights

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 83% found GrindCare effective.

The user-friendly GrindCare device

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)

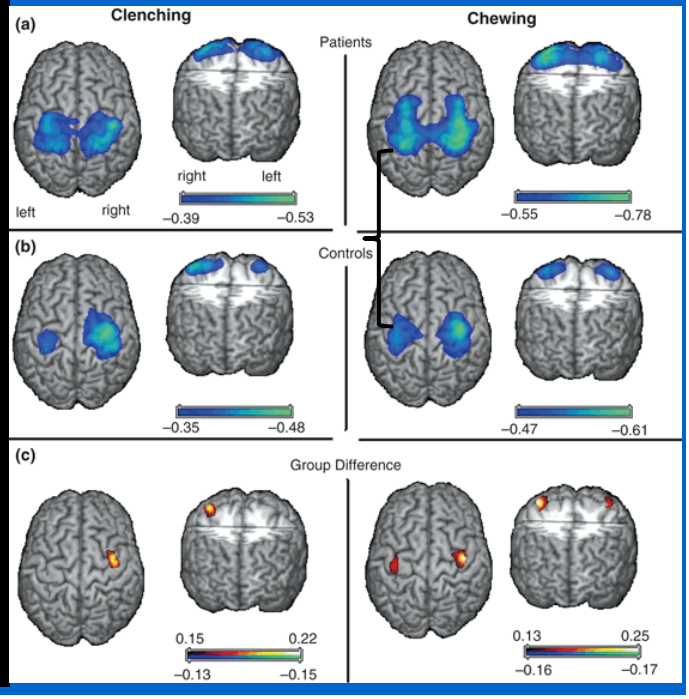
			No Bruxism		Bruxism			
		N	% (CI 95%)	N	% (CI 95%)	Total N	χ^2	*p*
OSAS	No OSAS	389	91.2 (85.8-93.6)	38	8.8 (6.4-14.2)	427	0	.93
	OSAS	180	91.0 (84.1-84.0)	18	9.0 (5.9-16.0)	198		
Snoring	No snoring	328	91.8 (89.0-95.5)	29	8.2 (4.5-11.0)	357	0.7	.39
	Snoring (3x or more/wk)	241	90.0 (81.1-92.1)	27	10.0 (7.9-18.9)	268		
RLS	No RLS	441	90.7 (86.5-93.2)	42	9.3 (6.8-13.5)	483	3.0	.38
	RLS	101	88.5 (60.8-97.6)	13	11.5 (2.4-39.2)	114		
Insomnia	No insomnia	508	92.0 (87.2-94.5)	44	8.0 (5.5-12.8)	552	5.6	**.01**
	Insomnia	61	83.5 (93.6-82.9)	12	16.5 (6.4-17.1)	73		
Anxiety	No anxiety	482	92.6 (90.1-95.1)	38	7.4 (4.9-9.9)	520	1.1	.28
	Anxiety	37	88.0 (69.7-94.3)	5	12.0 (5.7-30.3)	42		
Depression	No depression	468	92.6 (90.0-94.7)	37	7.4 (5.3-10.0)	505	1.0	.30
	Depression	47	88.6 (78.5-96.2)	6	11.4 (3.8-21.5)	53		

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

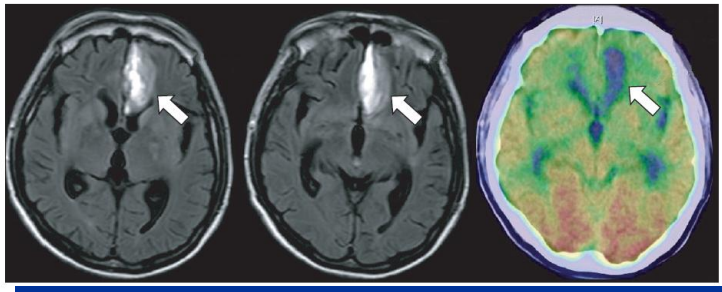


Fig. 1. Magnetic resonance imaging of the brain from case 1, showing subacute intracranial hemorrhage in the left basal frontal lobe (arrows). Positron emission tomography also shows metabolic defects in the left basal frontal lobe along with diffuse hypometabolism in the bilateral frontal, temporal, and parietal cortex.

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)

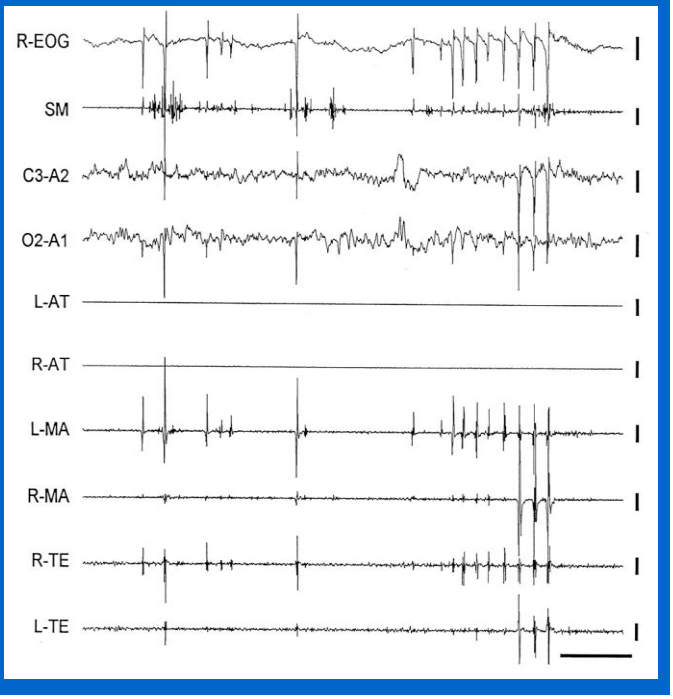
- Epilepsy (Vetrugno R 2002), Parkinson's, Huntingtons, Oromandibular dystonia or Neuroleptic induced dyskinesia……

Tooth Tapping

= Sleep oromandibular or orofacial myoclonus (found in 10% of SB patients)

Important to *EXCLUDE SLEEP EPILEPSIA*

(Kato T et al, Mov Disorders 1999; Vetrugno R et al, Familial nocturnal facio-mandibular myoclonus mimicking sleep bruxism. Neurology 2002)



AGAIN SB and Concomitant Neurological-Movement sleep disorders:

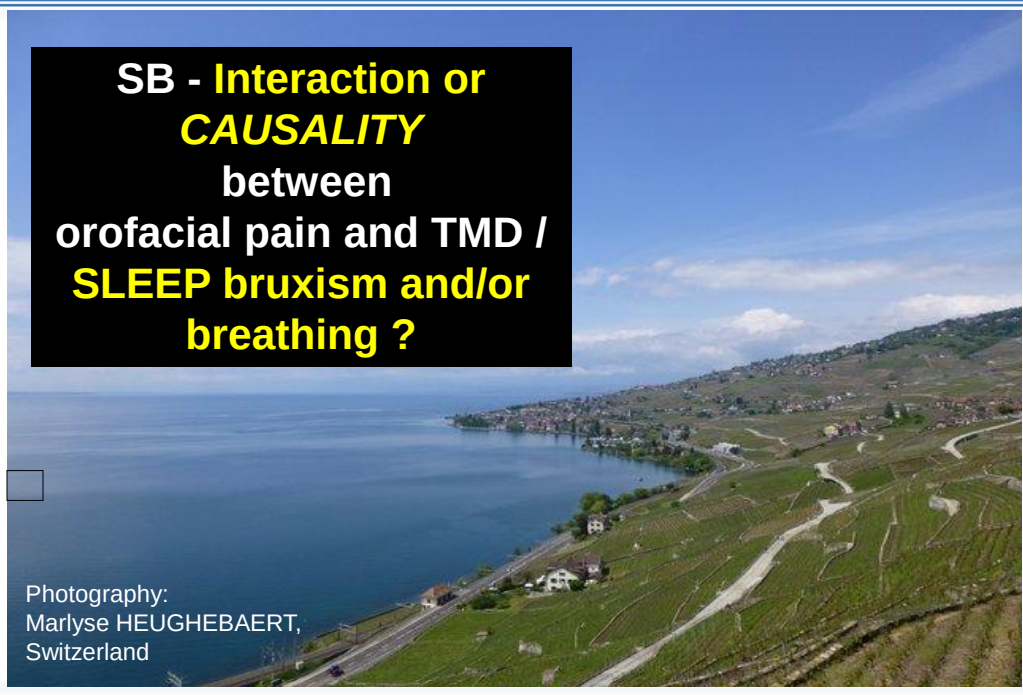
Periodic Limb Movement Syndrome

Mostly leg, about 40% in arm (+ 10 mvt/hr)

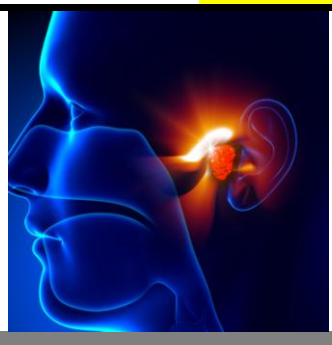
- **RLS (the wake variant of PLMS) and SB= 10% overlap** (pop survey, Lavigne & Montplaisir 1994)

- **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)



Temporomandibular Disorder and Morning Transient OroFacial Pain



More Sleep Bruxism EMG activity does not = More PAIN

TMD/Ctl = Same RMMA-SB index / K Raphael-JADA 2012

Laboratory polysomnographic (PSG) comparison of sleep bruxism (SB) among case and control participants.

PSG	CONTROL PARTICIPANTS (n = 46)		CASE PARTICIPANTS (n = 124)		*P* VALUE (χ^2 OR FISHER EXACT TEST)
Criterion	**No. (%)**		**No. (%)**		
Met research diagnostic criteria (RDC)/SB criteria	5 (10.9)		12 (9.7)		.818
Met subthreshold RDC/SB criteria	8 (17.4)		21 (16.9)		.915
Had two or more episodes with grinding noise	36 (78.3)		74 (59.7)		.038
Measure	**Mean (SD*)**	**Median**	**Mean (SD)**	**Median**	***P* Value (Median Test)**
RMMA episodes					
Count per hour	1.7 (1.9)	1.0	1.5 (1.9)	0.8	.388
Duration, seconds	56.4 (69.5)	24.5	47.9 (69.7)	21	.989
RMMA episodes with grinding					
Count per hour	1.0 (1.1)	0.5	1.0 (1.5)	0.4	.615
Duration, seconds	36.2 (49.9)	15.5	31.3 (55.0)	11.0	.294
Other orofacial activities					
Count per hour	9.4 (7.2)	7.1	10.1 (8.5)	7.1	.863
Duration, seconds	122.5 (130.9)	87.0	127.8 (122.5)	87.0	.937
Other muscular activities					
Count per hour	6.1 (2.9)	5.8	6.5 (4.5)	5.1	.605
Duration, seconds	139.3 (70.3)	119.5	150.0 (104.7)	127.5	.937

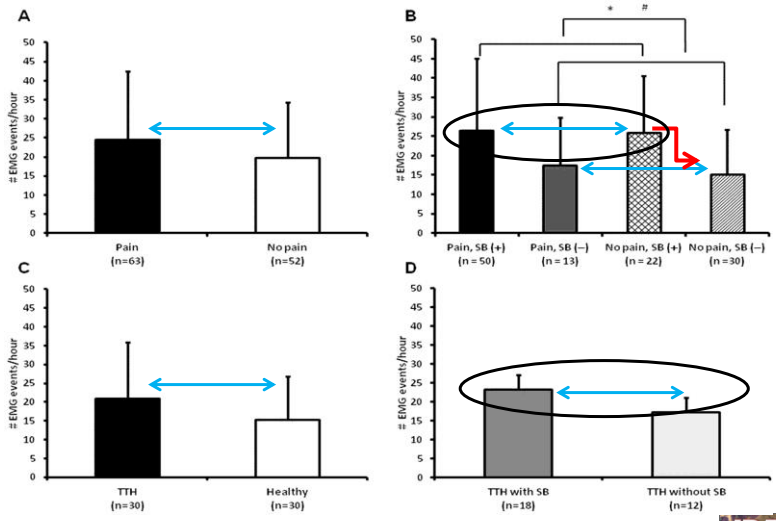
* SD: Standard deviation.

No more RMMA contraction in Morning Transient Pain (Abe S, JOFP, 2013)

Table 3 Sleep and RMMA Parameters for Controls (CTRL), Sleep Bruxers with Pain (SBrP), and Sleep Bruxers without Pain (SBrN)

	CTRL (n = 19) a	SBrP (n = 44) b	SBrN (n = 18) c	*P* value ANOVA†	Tukey test a vs b	Tukey test a vs c	Tukey test b vs c
Age	24.05 ± 1.26	26.27 ± 0.84	26.61 ± 1.31	.32			
Sex	8 F/11 M	30 F/14 M	9 F/9 M				
Sleep stage shift	**241.32 ± 15.35**	**197.80 ± 7.49**	**200.39 ± 11.71**	**.01**	**< .01**	**.02**	.86
Microarousals/hr*	9.33 ± 2.02	6.82 ± 0.62	7.38 ± 0.96	.64			
RMMA episodes							
Episodes/hr*	**1.34 ± 0.22**	**4.25 ± 0.42**	**5.15 ± 0.83**	**< .001**	**< .001**	**< .001**	.72
Phasic episodes/hr*	**1.00 ± 0.23**	**2.79 ± 0.38**	**2.99 ± 0.56**	**< .01**	**< .01**	**< .01**	.81
Tonic episodes/hr*	0.03 ± 0.02	0.11 ± 0.03	0.15 ± 0.09	.38			
Mixed episodes/hr*	**0.31 ± 0.09**	**1.28 ± 0.18**	**2.01 ± 0.42**	**.01**	**.15**	**.01**	.20
Episodes with noise*	**0.37 ± 0.17**	**7.18 ± 1.48**	**13.06 ± 4.46**	**< .001**	**< .001**	**< .001**	.64

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… ??

<u>Elevated - Sustained</u> 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 1st hr of sleep; also correlated to temporalis for all sleep duration (n=15 non pain subjects; Manfredini JOR 2011)

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

	Model 1 OR (95%CL)	Model 2 OR (95%CL)	Model 3 OR (95%CL)
High likelihood of obstructive sleep apnea [a]	3.48 (1.95, 6.19)	3.34 (1.87, 5.96)	3.63 (2.03, 6.52)

Differential Dx critical -
Concomitant sleep disorders breathing:

QUESTIONNAIRE and Sleep Recording:
See next slides

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

Table 1 – Mean and SD values for Sleep Architecture and Continuity, Respiratory and PLM Variables in TMD patients and Controls (Night 2 data, except for 10 participants whose Night 1 data were used, as described in the Method section).

	TMD Patients (n=124)		Controls (n=46)		p value*
PSG Parameters	**Mean**	**SD**	**Mean**	**SD**	
Sleep Continuity					
Total Sleep Time (TST, min)	386.4	53.1	402.3	45.8	0.139
Sleep Efficiency (SE) = TST/TRTx100%	89.7	8.7	92.3	6.4	0.128
Sleep Onset Latency (SOL) (min)	11.6	16.5	9.0	10.1	0.276
Number of Awakenings	18.2	11.3	14.3	7.8	0.069
Sleep Architecture					
N1 as a % of TST	12.2	7.6	9.2	5.0	0.034
N2 as a % of TST	51.9	10.6	51.1	10.3	0.898
N3 as a % of TST	16.5	10.9	19.0	8.5	0.356
REM as % of TST	19.3	6.9	20.6	5.7	0.324
REM Latency (min)	109.1	64.3	88.6	52.1	0.047
Arousals					
Spontaneous Arousal Index	10.6	6.0	12.0	5.6	0.195
Respiratory ArousalIndex	6.0	6.1	3.5	3.3	0.021
PLM Arousal Index	1.1	2.4	1.6	3.3	0.295
Total Arousal Index	17.8	8.2	17.0	6.8	0.855
Respiratory variables					
Apnea-hypopnea index (AHI)	3.7	6.6	2.4	3.9	0.504
Respiratory disturbance index (RDI)	8.1	8.5	5.0	5.1	0.056
RERA index total	4.3	4.3	2.6	2.7	0.017
PLM Index	4.9	10.3	5.8	13.8	0.424

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

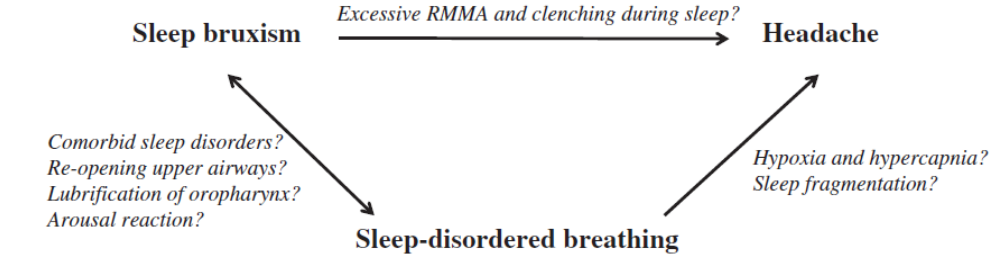


Fig. 3. Comorbid SB, headache, and SDB: putative mechanisms.

Carra, Dent Cli North Am 2012

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

Diagnoses	Male (n=10)		Female (n=43)		Total (N=53)	
	n	%	n	%	n	%
INSOMNIAS						
Psychophysiologic	2	20	9	20.9	11	20.8
Idiopathic	0	-	3	7	3	5.7
Primary Insomnia (DSM-IV-TR) (Includes psychophysiologic+idiopathic)	2	20	12	27.9	14	26.4
Insomnia due to TMD	0	-	3	7	3	5.7
Insomnia due to Mood Disorder	0	-	2	4.7	2	3.8
Any Insomnia Diagnosis	2	20	17	39.5	19	35.8
OBSTRUCTIVE SLEEP APNEA						
Mild (RDI = 5-14.9)	2	20	9	20.9	11	20.8
Moderate (RDI = 15-29.9)	2	20	0	-	2	3.8
Severe (RDI ≥ 30)	1	10	1	2.3	2	3.8
Any Sleep Apnea DX	5	50	10	23.2	15	28.4
MOVEMENT DISORDERS						

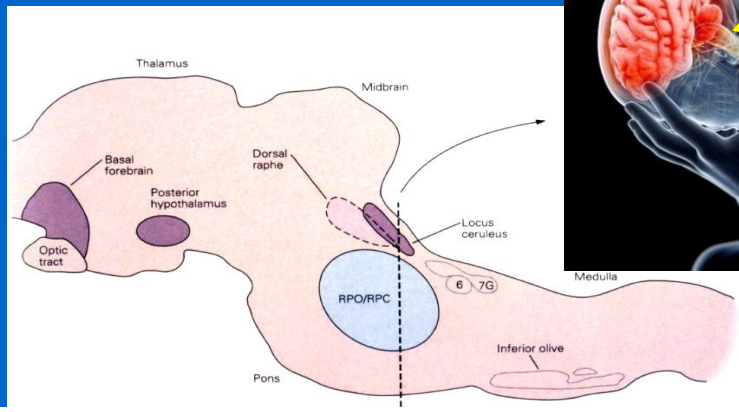
Sleep Lab, TMD population

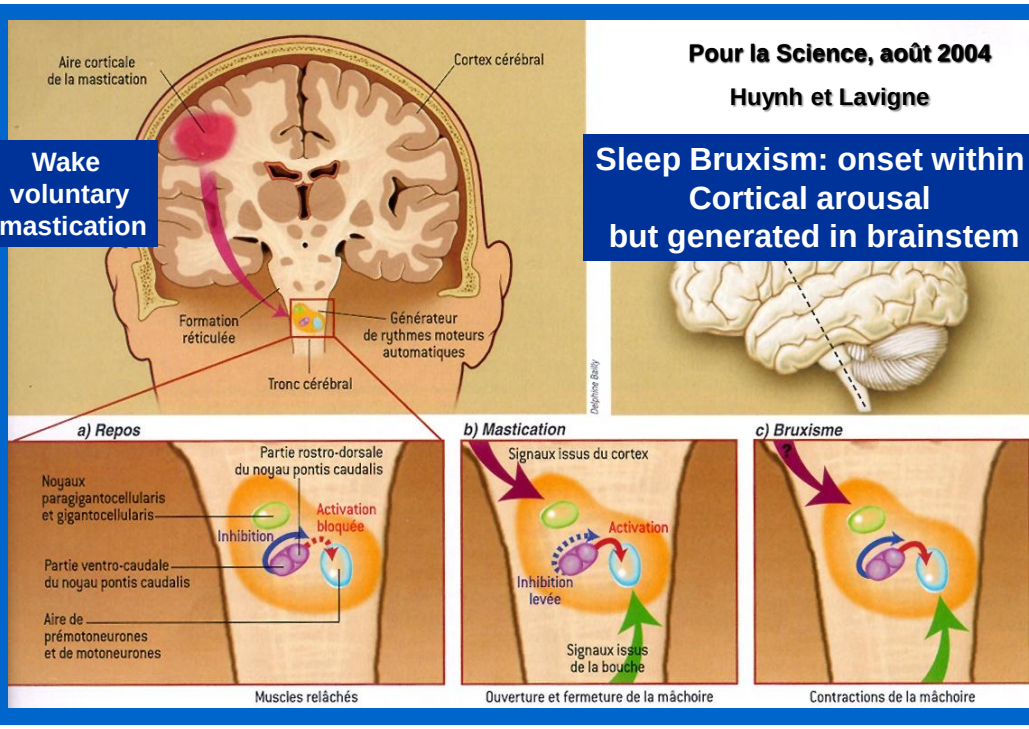
- **35.8% INSOMNIA**
- **28.4% OSA**
- **17.3% SLEEP BRUXISM**

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





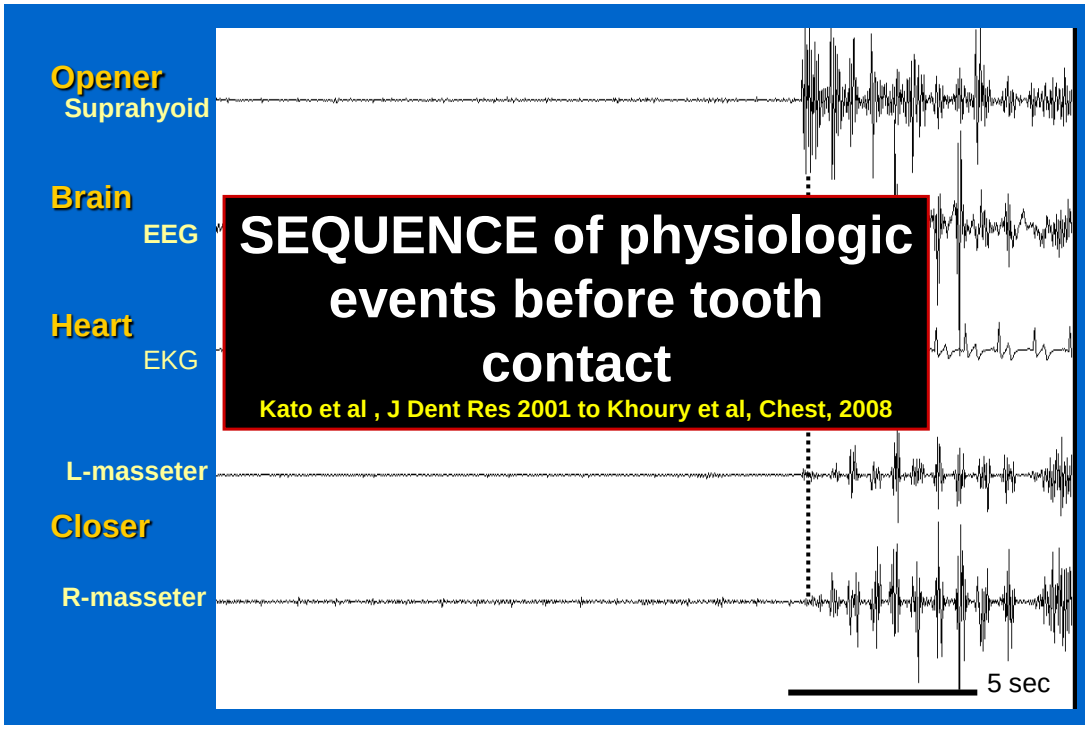
Sleep arousal (natural mechanism)

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

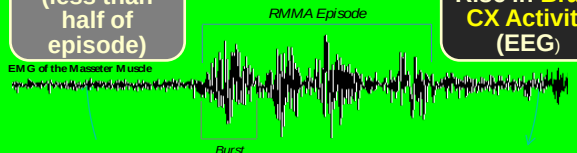


Sequence of Physiological Activities Associated to RMMA-SB Episodes in PRESENCE of Sleep Arousal (observed with 50-80% of Episodes)

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

Swallowing
(less than half of episode)

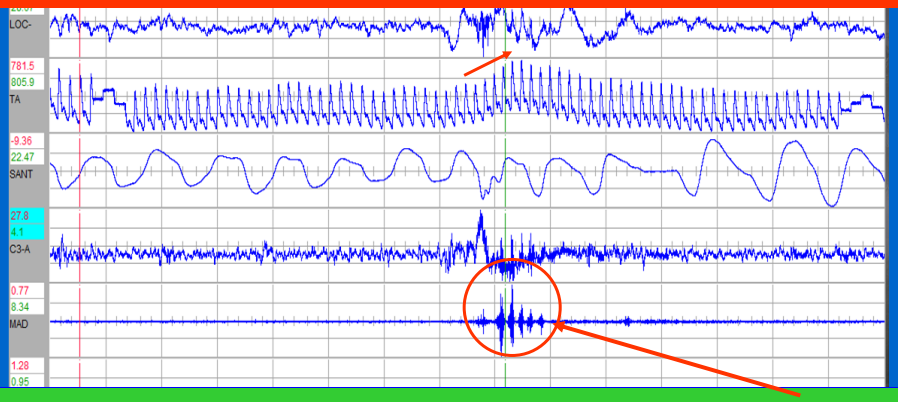
- 4 sec:
Rise in Brain CX Activity (EEG)



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

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

Risk if already Hx high blood pressure is UNKNOWN



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

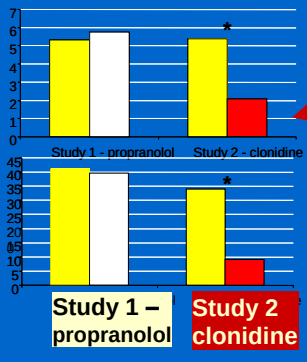
Awarded by SLEEP – APSS meeting 2013

The Challenge – proof of concept

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

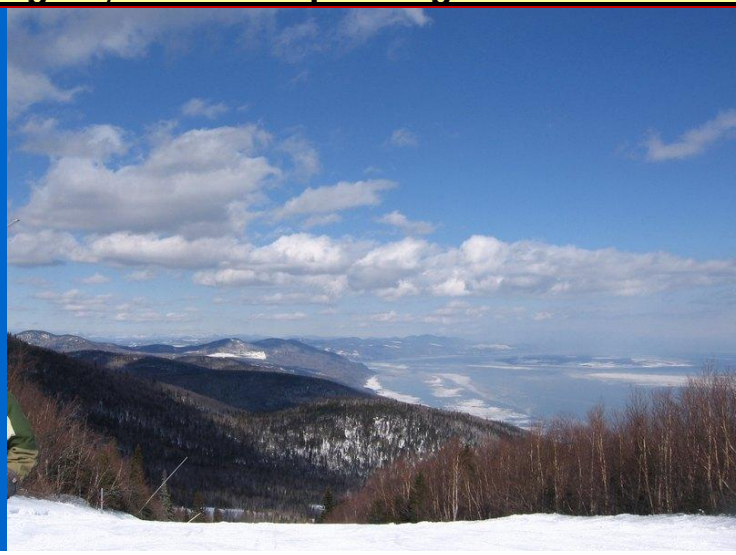
***2- Clonidine 0.3 mg*:**

60% reduction but hypotension in 20% of subjects (Huynh et al, SLEEP 2006)

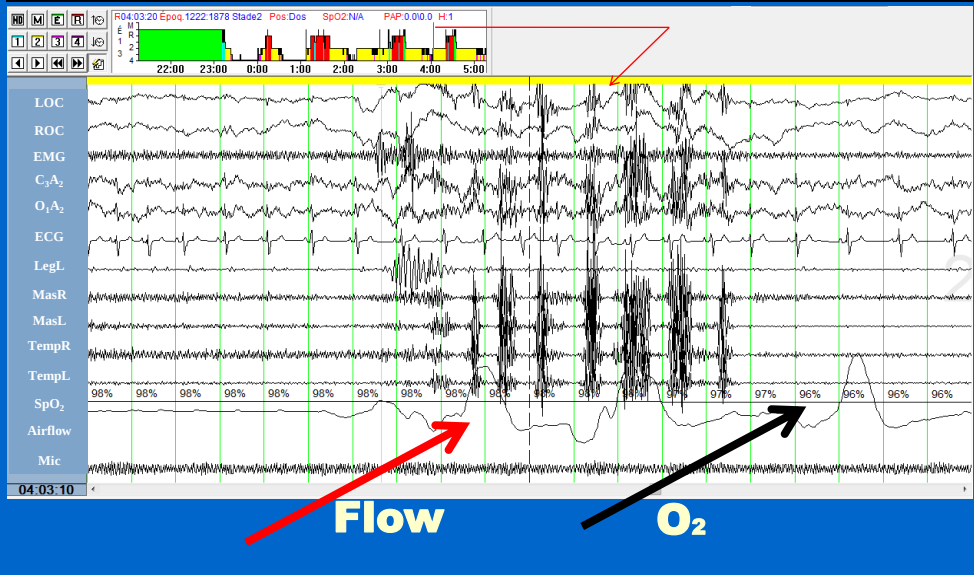
Unpublished data coming from Baba, Japan with 0.1 mg

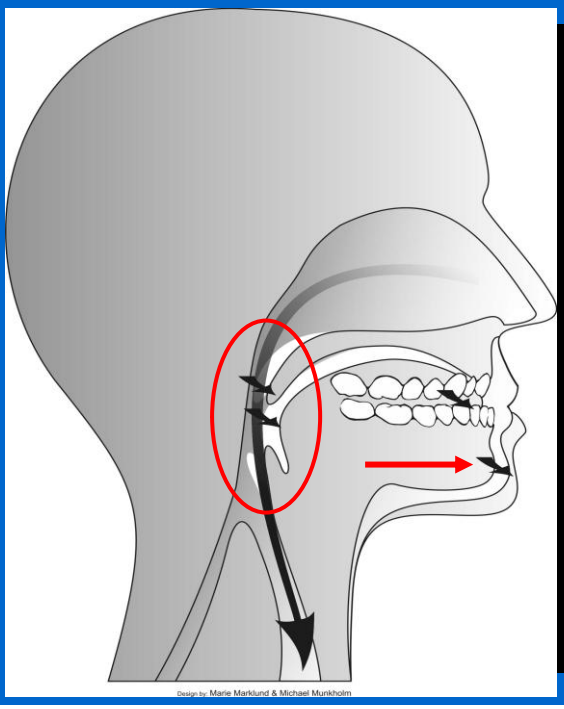
SB- *FOCUS on airway & respiration*: May be related in some patient?

Again, it is not explaining all causes of SB



RMMA and BREATHING : Flow and Oxymetry (O_2)





Airway in sleep: tongue and mandible tend to move downward and backward

Oropharyngeal domain…

Cistulli et al Sydney, Australia

Sleep disorders breathing crescendo

Increasing upper airway collapsibility

- Occasional snoring
- Habitual snoring

TO

- Upper airway resistance syndrome-
- RERA= Respiratory Event Related Arousal

TO

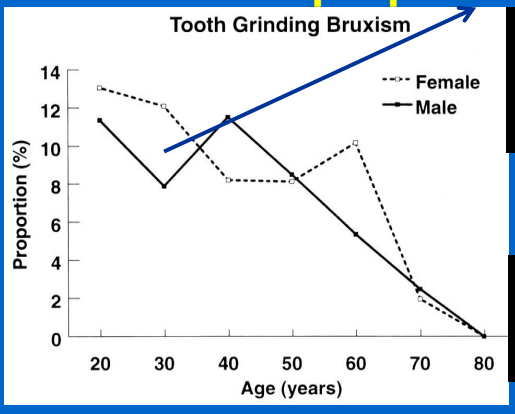
- Occasional apneas or hypopneas
- Obstructive Sleep Apnea (OSA) syndrome

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)

AHI	No bruxism	569	7.7 (4.4-11.1) ←	12.44	.31
	Bruxism	56	6.2 (6.8-8.9)	10.09	
SaO_2 mean (%)	No bruxism	569	95.2 (94.9-95.9) ←	1.92	.38
	Bruxism	56	95.4 (95.0-95.4)	1.52	

JDR JOURNAL OF DENTAL RESEARCH

Temporal association

Cause and effect:

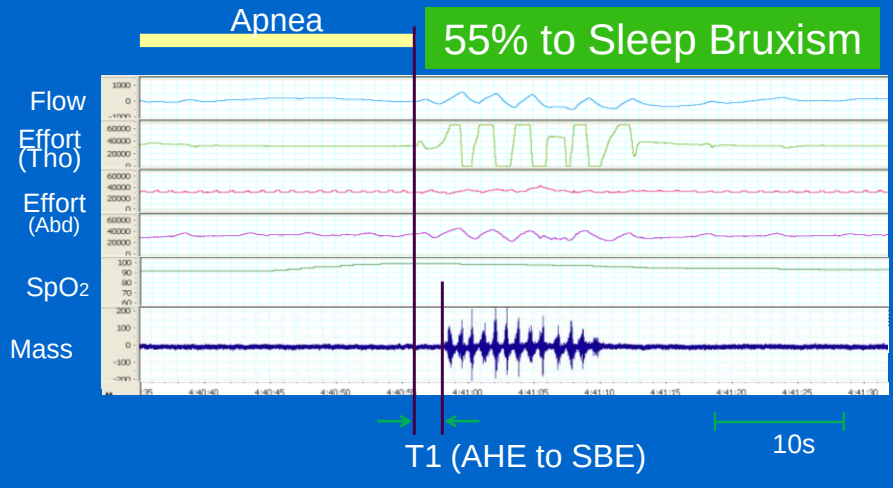
Cause should precede the effect

- **RMMA and Apnea or hyponea timing**
- **Can be experimentally reproduced**

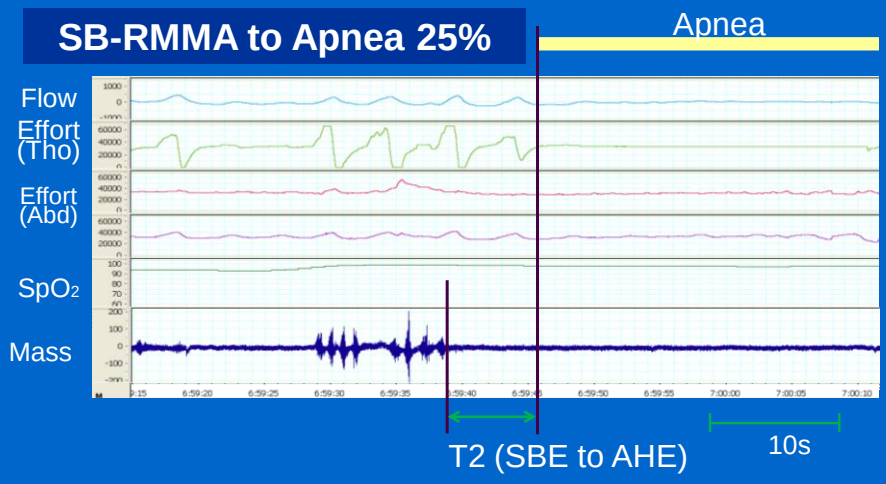
Or

Altered by treatment

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



SB-RMMA to Apnea 25%



20% of SBE not associated to Apnea events

The Challenge – proof of concept

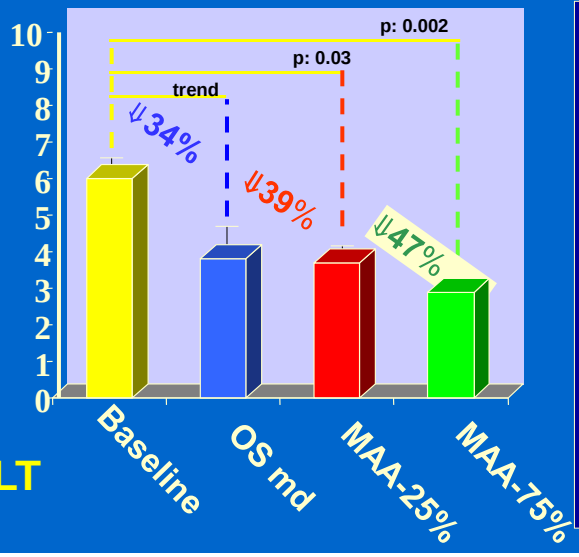
Opening airway reduce RMMA

see next slides

NOT ALL…

Lower # RMMA episodes/hr with occlusal (bite) splint (lower) and a MAA (Silencer, BC)

(A. Landry-Schonbeck, Int J Prostho 2009)



ADULT

Mild benefit For SB patients

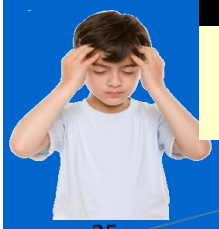
No fracture of MAA

SHORT TERM

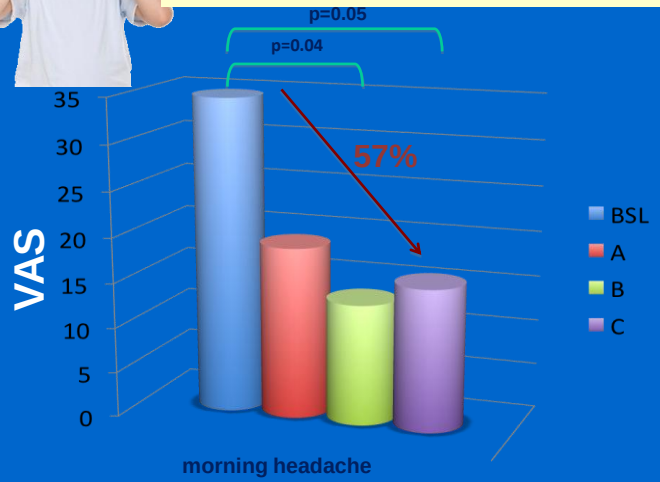
MC CARRA – SLEEP MEDICINE 2013

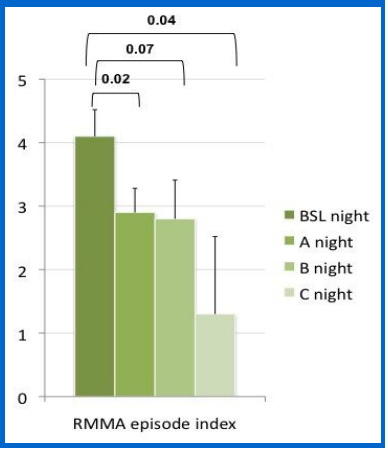
OFF LABEL / NOT FDA – PRE ORTHODONTIC PATIENTS

Less Headache in the morning Lower RMMA index



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





Clinical association is not causality

Diagnoses	Male (n=10)		Female (n=43)		Total (N=53)	
	n	%	n	%	n	%
INSOMNIAS						
Psychophysiologic	2	20	9	20.9	11	20.8
Idiopathic	0	-	3	7	3	5.7
Primary Insomnia (DSM-IV-TR)						
(Includes psychophysiologic+idiopathic)	2	20	12	27.9	14	26.4
Insomnia due to TMD	0	-	3	7	3	5.7
Insomnia due to Mood Disorder	0	-	2	4.7	2	3.8
Any Insomnia Diagnosis	2	20	17	39.5	19	35.8
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Any Sleep Apnea DX	5	50	10	23.2	15	28.4
MOVEMENT DISORDERS						

Sleep Lab, TMD population & COMORBIDITIES SMITH, SLEEP 2009

- **35.8% INSOMNIA**
- **28.4% OSA**
- **17.3% SLEEP BRUXISM**
  - **45% of TMD patients 1 sleep disorder**
  - **26% of TMD patients 2 sleep disorder**
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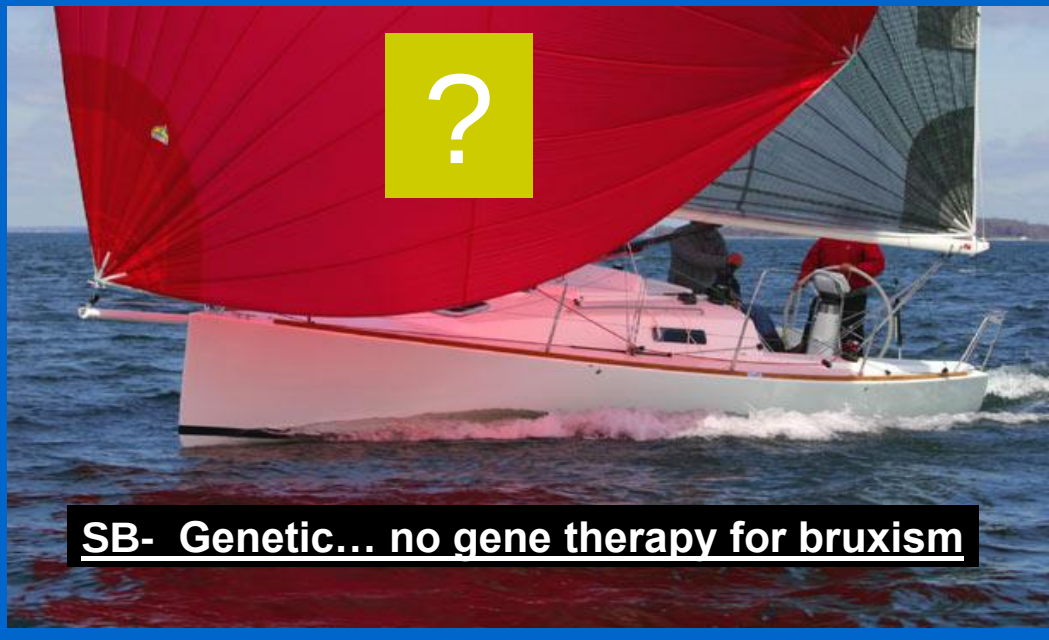


Phenotyping SB patients and blood relatives for genetics polymorphism

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

Phenotyping SB patients and blood relatives for genetics polymorphism

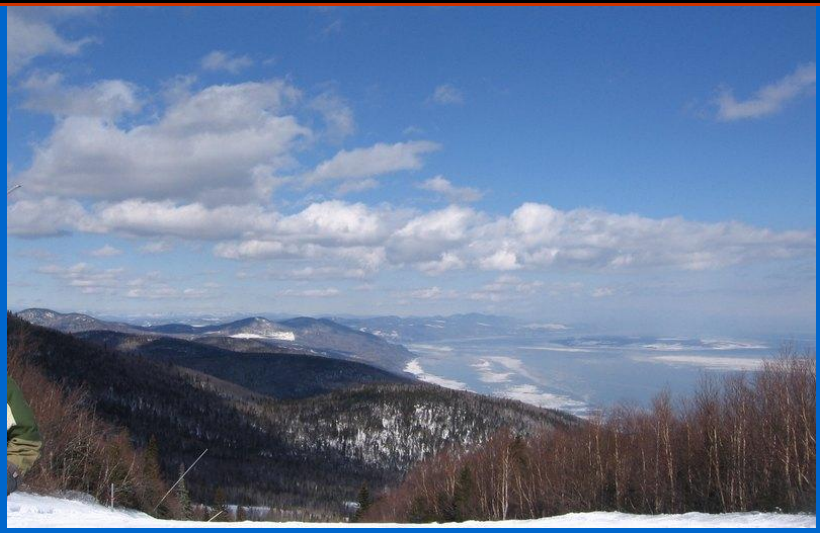
SLEEP LAB STUDY:

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

SB- Management
(not Tx, please be realistic)



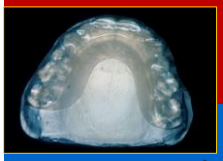
VARIOUS management for SB: *Effect & Level of evidences*
Winocur, in Sleep Med for Dentist, Quintessence,2009

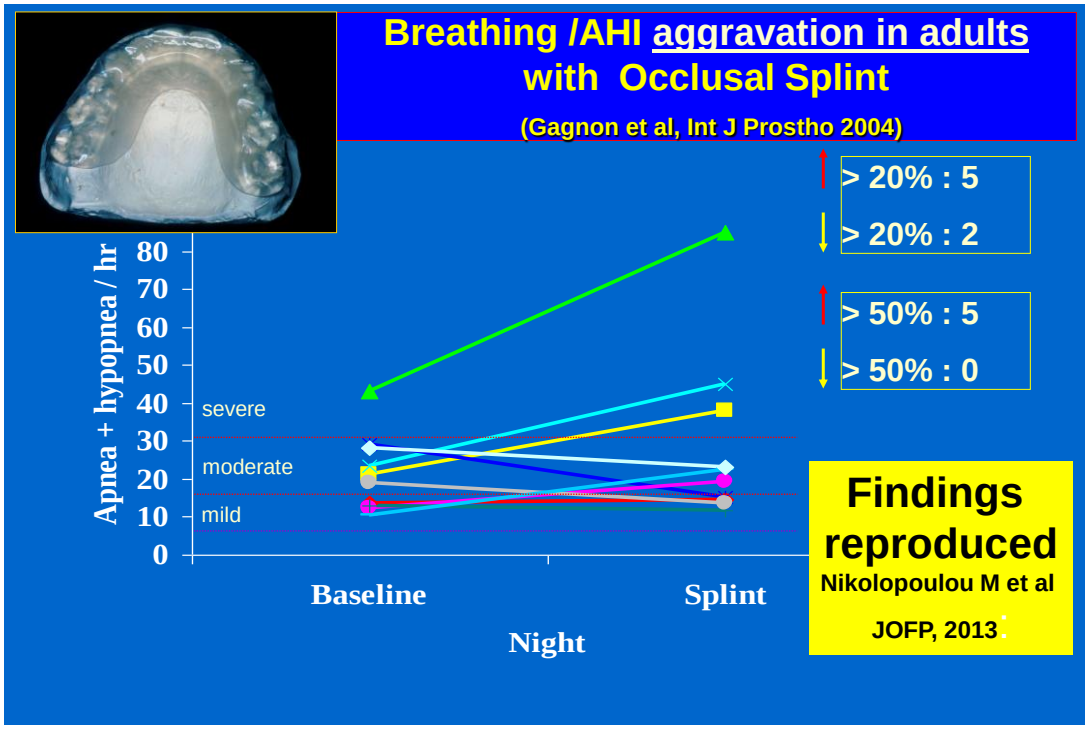
Behavioral management approaches:

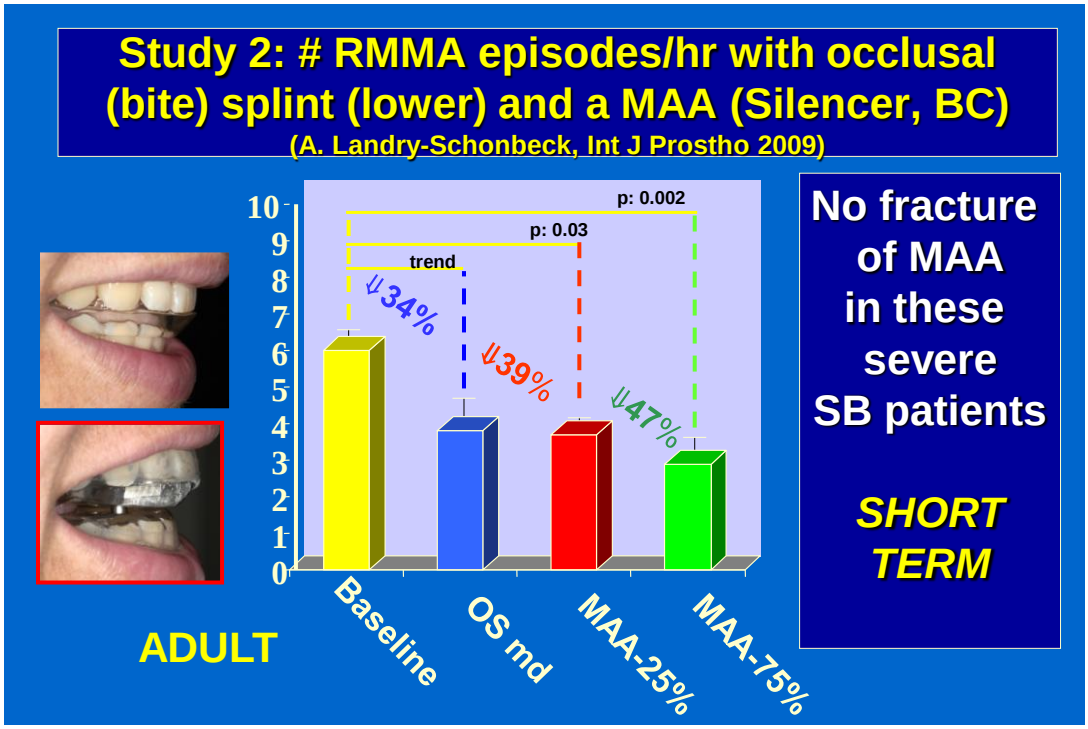
- **Explanation of causes and exacerbation factors for SB**
- **Elimination of clenching teeth and bracing jaw during daytime in reaction to life pressures**
- **Lifestyle changes; introduction of sleep hygiene, relaxation, autohypnosis, and winding down before sleep**
- **Physical therapy and training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

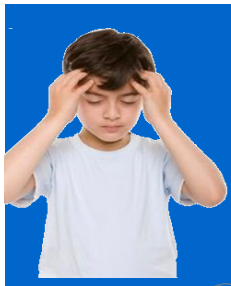
Questionable effect – Weak evidence so far but patient report subjective well being!

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




Breathing /AHI aggravation in adults with Occlusal Splint
(Gagnon et al, Int J Prostho 2004)
> 20% : 5
> 20% : 2
> 50% : 5
> 50% : 0
Apnea + hypopnea / hr
80
70
60
50
40
30
20
10
0
severe
moderate
mild
Baseline
Splint
Night
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)
p: 0.002
p: 0.03
trend
⇓34%
⇓39%
⇓47%
10
9
8
7
6
5
4
3
2
1
0
Baseline
OS md
MAA-25%
MAA-75%
ADULT
No fracture of MAA in these severe SB patients
SHORT TERM

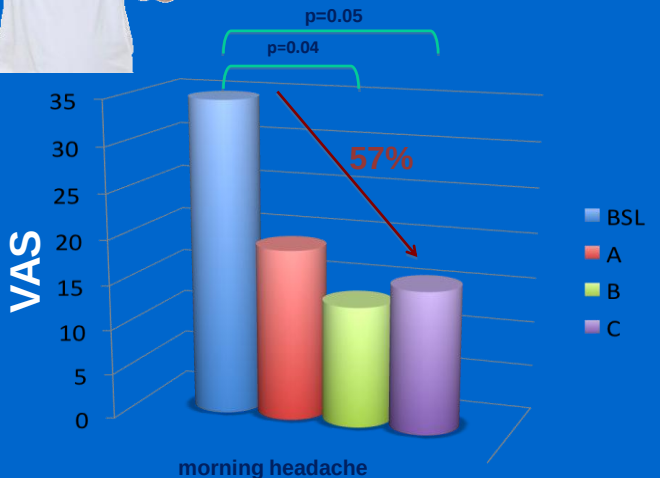


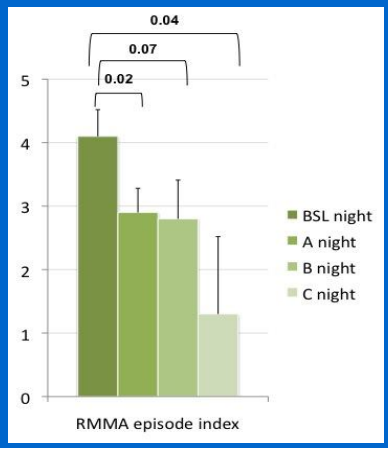
MC CARRA – SLEEP MEDICINE 2013 ----- OFF LABEL / NOT FDA

Frequent headache (as a complaint)

➢ **Headache in the morning**

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





OVERVIEW on PHARMACOLOGICAL management for SB

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
– Propanolol= No effect in RCET

NEW RESEARCH

JCSM
Journal of Clinical Sleep Medicine
http://dx.doi.org/10.5664/jcsm.3532

Effects of Botulinum Toxin on Jaw Motor Events during Sleep in Sleep Bruxism Patients: A Polysomnographic Evaluation

Young Joo Shim, D.D.S, M.S.D.[1]; Moon Kyu Lee, M.D., Ph.D.[2]; Takafumi Kato, D.D.S., Ph.D.[3]; Hyung Uk Park, D.D.S., M.S.D.[4]; Kyoung Heo, M.D., Ph.D.[5]; Seong Taek Kim, D.D.S., Ph.D.[6]

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

GrindCare Because better days start with good nights

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 83% found GrindCare effective.

The user-friendly GrindCare device

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

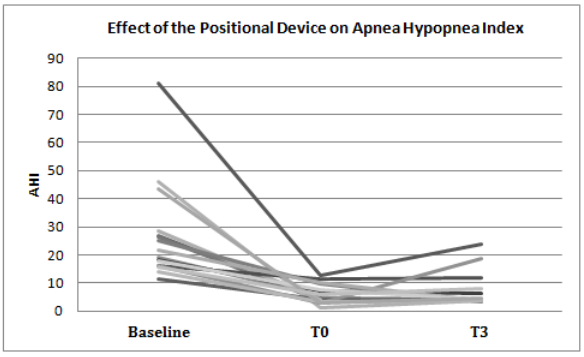
After controlling for 16 variables

Of occclusion= 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



Sleep Position Trainer

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

Yellow or Green Light

Absence of Sleep Disorder Breathing

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

Mandibular Advancement Appliance Or CPAP

With or without medication (see below)

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