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ORIGINAL ARTICLE

Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion

Daniele Manfredini[1] · **Luca Guarda-Nardini**[1] · **Rosario Marchese-Ragona**[2] · **Frank Lobbezoo**[3]

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Abstract
Background Sleep bruxism (SB) is a term covering different motor phenomena with various risk and etiological factors and potentially different clinical relevance, especially as far as its possible protective role against obstructive sleep apnea (OSA) is concerned. The present expert opinion discusses the possible temporal relationships between the two phenomena.
Methods Four hypothetical scenarios for a temporal relationship may be identified: (1) the two phenomena are unrelated; (2) the onset of the OSA event precedes the onset of the SB event within a limited time span, with SB having a potential OSA-protective role; (3) the onset of the SB event precedes the onset of the OSA event within a limited time span, with SB having an OSA-inducing effect; and (4) the onset of the OSA and SB event occurs at the same moment.
Results Literature findings on the SB-OSA temporal relationship are inconclusive. The most plausible hypothesis is that the above scenarios are all actually possible and that the relative predominance of one specific sequence of events varies at the individual level. SB activity may be protective against OSA by protruding the mandible and restoring airway patency in those subjects who benefit from mandibular advancement strategies or may even be related to OSA induction, as a consequence of airways' mucosae swelling resulting from a SB-induced trigeminal cardiac reflex.
Conclusions Clinicians should keep in mind that the SB-OSA relationship is complex and that interindividual differences may explain the possible different SB-OSA relationships, with particular regard to the anatomical site of obstruction.

Keywords Sleep bruxism · Bruxism · Obstructive sleep apnea · Relationship

Introduction

Bruxism is an oromandibular condition characterized by different activities of the jaw muscles (i.e., teeth clenching or grinding, mandible thrusting) and circadian manifestations (i.e., sleep bruxism (SB) or awake bruxism (AB)) [1]. In recent years, efforts were made to get a deeper insight into the pathophysiology and clinical relevance of the different bruxism activities, especially as far as the observation is concerned that some bruxism phenomena occurring during sleep are not necessarily the expression of a pathological condition [2–5].

Obstructive sleep apnea (OSA) is a primary sleep disorder related to collapse of the upper airways, which may cause total (i.e., apnea) or partial (i.e., hypopnea) obstruction and may lead to oxygen desaturation and arousal, viz., awakening from sleep [6]. As in the case of SB, the pathophysiology of OSA is not yet fully understood, with a combination of neuromuscular and anatomical factors playing a role in the pathogenesis of obstructions [7].

Both SB and OSA are related with sleep arousal episodes, viz., micro-awakenings featuring physiological activations

Daniele Manfredini holds DDS, PhD, University of Padova.

✉ Daniele Manfredini
daniele.manfredini@tin.it

[1] Temporomandibular Disorders Clinic, Department of Maxillofacial Surgery, University of Padova, Padova, Italy

[2] Institute of Otolaryngology, Department of Neurosciences, University of Padova, Padova, Italy

[3] Department of Oral Kinesiology, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University Amsterdam, MOVE Research Institute Amsterdam, Amsterdam, The Netherlands

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[3], and OSA prevalence seems to be higher than in general population, being reported in more than 30 % of adults with possible SB [8]. A clinical relationship between the two phenomena was suggested by some early investigations describing a tonic or phasic activity of the masseter muscle satisfying the criteria for SB at the end of the apneic event [9–13]. Based on that, a possible protective role of SB against OSA was suggested [4]. Notwithstanding that, the available evidence is not enough to even confirm or deny the association between the two phenomena [14].

Materials and methods

Considering these premises, this expert opinion deals with the above topic by analyzing the different temporal relationships that are theoretically possible between SB and OSA, viz., does bruxism-like jaw muscle activity actually follow or precede the apnea, do they coincide, or are the events temporally unrelated?

Results

In theory, the temporal relationship between SB and OSA events may lie within one of the following possibilities:

– Scenario no. 1: The two phenomena are unrelated, as far as their temporal sequence is concerned

In this scenario, polysomnography (PSG) data shows the temporally unrelated occurrence of SB and OSA events, viz., the two phenomena do not occur both within a limited time span, suggesting that the two conditions may be a part, for instance, of different spectra of sleep arousals (Fig. 1). At present, this hypothesis is not supported by any current evidence due to the paucity of studies specifically designed to assess this issue. Moreover, there is no consensus on the time lag that should be set as threshold to consider the two events as being related. The only available paper on the topic set an arbitrary cutoff at a 5-min span to hypothesize that SB and OSA events occurring during that interval are linked to each other [13]. In the clinical setting, examples of this scenario refer to any idiopathic SB event or to those SB episodes that are secondary to drugs or neurological diseases and have nothing to do with breathing disorders, but more research is needed to better define the time issue and standardize strategies of data collection on this topic. Also, an improvement in knowledge on the pathophysiology of SB and OSA with respect to clinical meaning of sleep arousals (i.e., different intensity levels and physiological origin) is fundamental to verify the actual relevance of this scenario [15].

– Scenario no. 2: The onset of OSA precedes the onset of SB

In this scenario, OSA events precede SB (Fig. 2). In some cases, the apnea seems to be interrupted by a contraction of the masseter muscles. PSG data identify bursts of masseter activity that may satisfy the criteria for a SB episode (i.e., bursts with an EMG amplitude twice higher than baseline activity and a duration longer than 0.25 s) at the end of the apnea event. In the cascade of arousal events, an activation of the suprahyoid muscles was described in the second before the SB event [16]. Such a muscle activation may have the scope of protruding the mandible to try restoring the airway patency, in accordance with the hypothesis that a rise in the suprahyoid muscle activity concurs with the airways opening in SB subjects [3] as well as with observations that the return of ventilation in OSA patients and an increase in the respiratory pattern are associated with a rise in suprahyoid and genioglossal muscle tone [17, 18]. PSG data show that oxygen saturation quickly increases back to physiological levels in a few seconds after the SB event.

This kind of temporal relationship provides a possible example of a physiological role of SB events. Based on this potential protective role against OSA, the recent consensus definition for SB abandoned the negative term “disorder” in favor of the more neutral word “condition” to indicate bruxism [1].

A potential confirmation of such possible protective role of SB came also from recent observations that mandibular advancement devices (MAD) may be useful to reduce both SB and OSA events, with up to a 60 % reduction of SB events in OSA patients wearing MAD [19–21]. Given these premises, an interesting speculation is that the possible protective role of jaw muscle activities depends on the type and anatomical location of the obstruction. Indeed, OSA is a multifaceted disorder with different potential causes and anatomical sites of airway collapse. The latter should be carefully identified by means of sleep nose endoscopy (SNE), which allows identifying those subjects who could have their obstruction solved with mandibular traction [22]. This may suggest that the hypothesis of a goal-oriented muscle activation at the end of an OSA event is biologically plausible only in those subjects with an anatomical obstruction that is solved with an advancement of the mandible. On the contrary, an individual with apnea events due to an obstruction that cannot be solved with mandibular traction (i.e., collapse of the airways at the

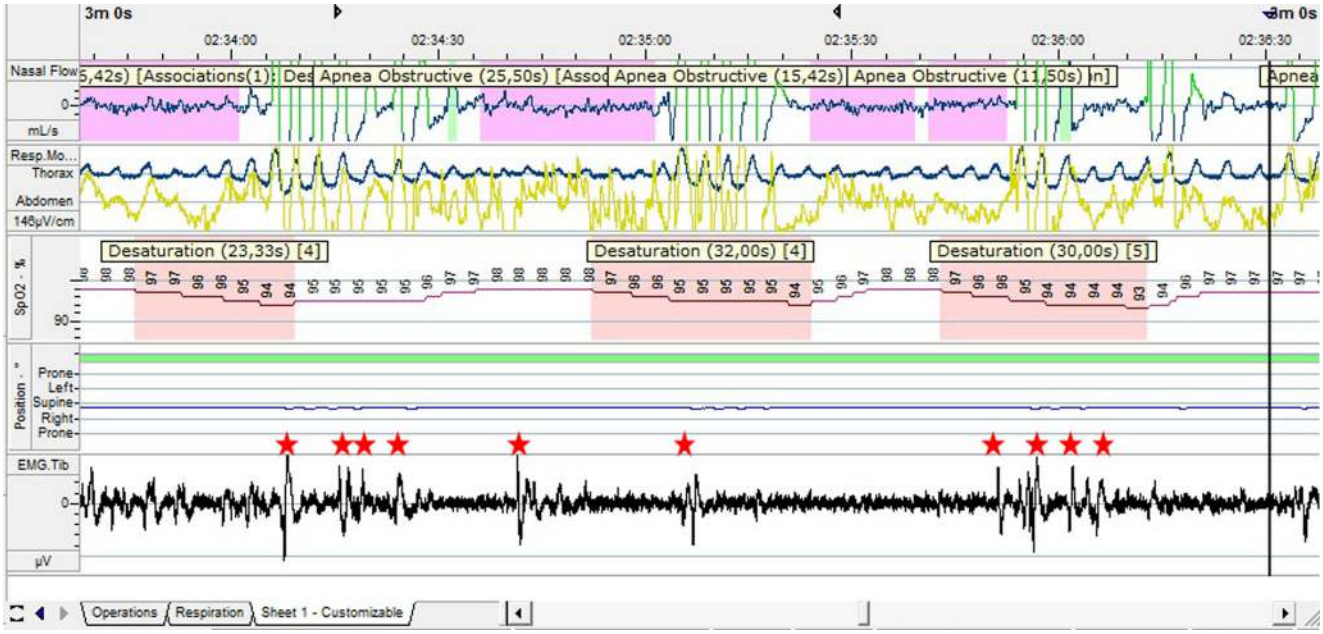


Fig. 1 PSG recording over a 3-min epoch length of a 58-year-old male with moderate OSA (AHI 18.7). The right masseter muscle EMG activity is shown in the tibialis channel (*black trace*). Apnea (*violet areas*) events are evidenced in the nasal flow channel at the *top of the screen*. Peaks of increased masseter EMG activity are evidenced with *red flashes above the black trace* of the tibialis channel. OSA events and masseter activity are not temporally related

lower pharyngeal levels) might be less likely to show a protective bruxism-related muscle activation aiming to restore airway patency. Future studies assessing this hypothesis with respect to the possibility that a physiological response of mandibular protrusion to protect the airway could hypothetically occur regardless of the site of obstruction are recommended.

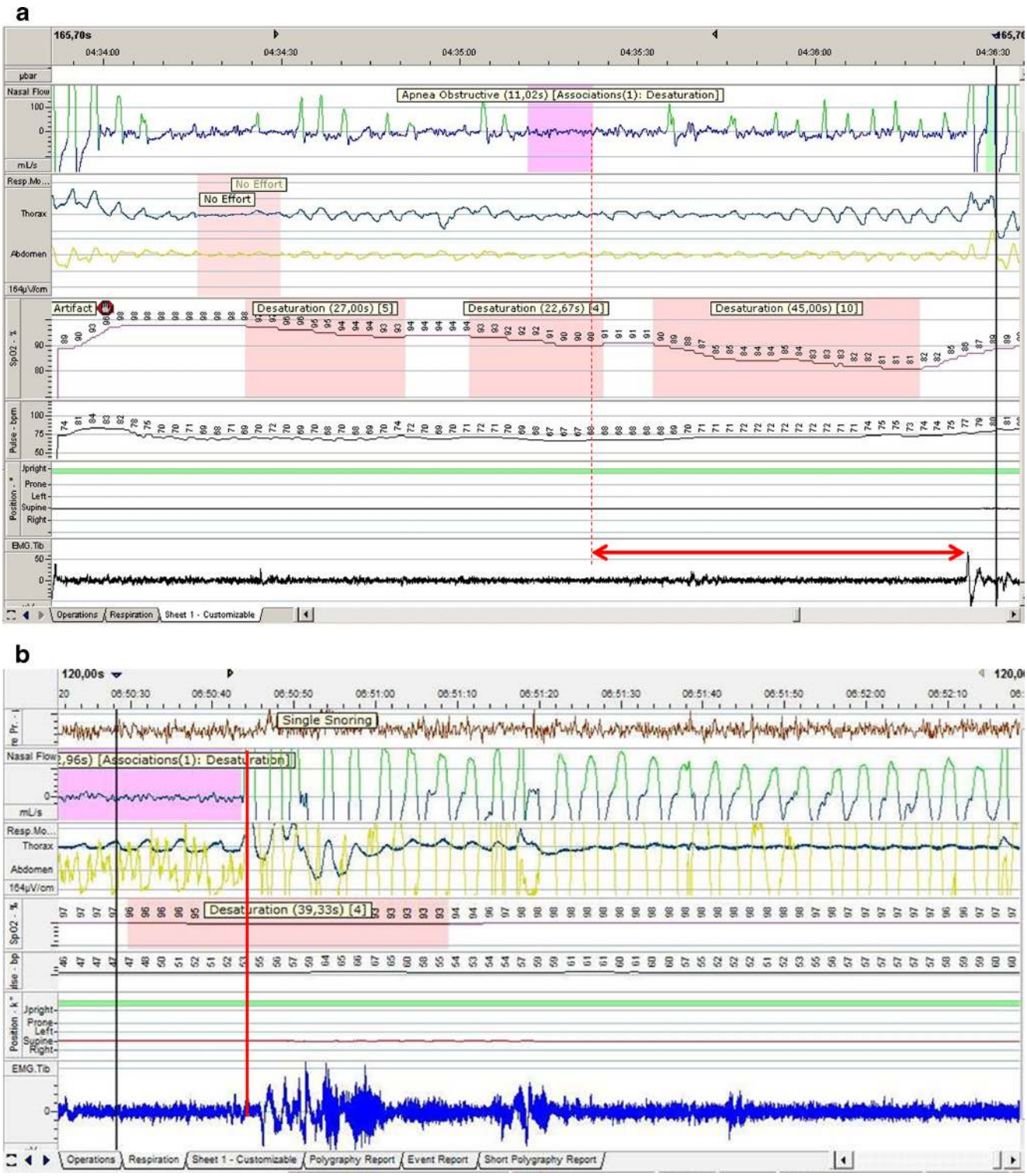
In addition, it should also be noted that the masseter muscle contraction associated with airway obstruction may be a non-specific muscle reaction, as this has also been reported to occur in the anterior tibialis muscle [15]. Based on that, the possible occurrence of underlying disorders (e.g., unspecific upper airway resistance syndrome (UARS) without the occurrence of obstructive apneas and hypopneas) that may lead to respiratory effort-related arousals of sleep (RERAS) masking SB should be taken into account as well [23].

Within the framework of this second scenario, it is also noteworthy that, even in moderate or severe OSA patients, there are no apneic events during the sleep periods characterized by long-lasting tonic jaw muscle activities typical of jaw clenching. On the one hand, this observation may support the hypothesis that, along with phasic motor activities [24], also tonic SB-related muscle activities may protect against airway obstruction, possibly via different mechanisms. An interesting hypothesis to explain the potential clenching-related protection of the airway is the onset of a co-activation of oropharyngeal muscles, which then tense (i.e., improve their tone) due to the clenching activity, thereby reducing the apnea. Thus, on the other hand, the above described observation means that, given the evidence that clenching-type SB is likely related with stress sensitivity and anxiety traits [2, 25, 26], thus being a reactive phenomenon with a clear pathophysiology and clinical relevance, it may also yield a sort of secondary, not goal-oriented, prevention of OSA.

– Scenario no. 3: The onset of SB precedes the onset of OSA

Contrary to the above hypothesis, this scenario involves the possible initiating role that SB events may have with regard to apnea (Fig. 3). Even if more rarely described and not yet fully understood, this kind of temporal relationship may find support in the otolaryngology and sleep medicine literature suggesting that REM-sleep phases may be accompanied by mucosal swelling of the upper airways, leading to nasal congestion [27]. Such nasal congestion recognizes its pathogenesis in a trigeminal activation and may be responsible for apnea in those subjects having REM-predominant OSA events [28].

Support to this scenario may also come from the studies on the physiology of sleep arousals, which are centrally mediated



"micro-awakenings" featuring several sequential changes in some physiological parameters. Arousal phenomena have a complex relationship with both SB and OSA [3]. In general, it is commonly accepted that such arousals are characterized by a transient electroencephalographic activity as well as an increase in heart rate and blood pressure, finally resulting in a SB event [3, 4]. A reduction of breathing amplitude may induce RERAS triggering the arousal [23]. Thus, under the most plausible scenario, an OSA event should precede SB, as described above. On the other hand, a number of arousals occur

◀ **Fig. 2** **a** PSG recording over a 3-min epoch length of a 75-year-old male with severe OSA (AHI 35.5). Channels are in Fig. 1a. An apnea (*violet areas*) event is evidenced in the nasal flow channel. A burst of marked EMG activity increase, satisfying the criteria for SB, follows OSA with about a 1-min delay, as evidenced by the *red line with arrows at the extremities*. The clinical meaning of this kind of relationship between OSA and SB events is yet to be understood, especially as far as the between-phenomena time span to consider them related is concerned. **b** PSG recording over a 2-min epoch span of a 54-year-old male with moderate OSA (AHI 21.2). The right masseter muscle EMG activity is shown in the tibialis channel (*blue trace*). The SB event, shown by the marked increase in masseter EMG activity, follows and seems to interrupt the OSA event (*red vertical line* was drawn to show the temporal proximity between the two events). Oxygen saturation progressively increases to normal levels within some seconds after the SB event. Interestingly, the masseter EMG activation is initially phasic in the first few seconds, and then becomes tonic in the second part of the SB episode. Two other less-intense episodes of tonic masseter activity are shown in this temporal sequence, with a progressive normalization of the EMG activity. **c** PSG recording of the same patient as in Fig. 2b. During this period of tonic, prolonged, clenching-type masseter activity, OSA events are not observed

without a respiratory trigger, and there is increasing evidence that the SB event following the arousal is associated with a trigeminal cardiac reflex activation which in turn leads to a reduction in heart rate [29, 30]. Based on that, SB may even be seen as a goal-oriented phenomenon aiming to restore bradycardia via the initiation of a trigeminal cardiac reflex which, in turn, may induce nasal congestion. The observation that clonidine, an anti-hypertension drug that lowers heart rate, may be effective to reduce SB events supports this hypothesis [31]. Thus, the possibility of an "inverted" temporal sequence of events with respect to the second scenario, viz., SB precedes apnea, cannot be disregarded. Also, in support of this hypothesis came a case report of a patient in which SB episodes preceded invariably catathrenia and bradypneic events [32] and, more relevantly, a recent paper on ten patients with OSA and SB, showing that one fourth of SB events precede an apneic event occurring within an arbitrarily set 5-min span [13].

Within these premises, it should be remarked that such intriguing hypothesis is in contrast with current knowledge about the distribution of SB episodes during the sleep cycle, with about 80 % of them occurring in nREM stages [3, 18, 24]. Thus, support to the plausibility of this scenario might be found only by studying the pathophysiology of the less common REM-SB events.

– Scenario no. 4: The onset of OSA and SB occurs at the same moment

In theory, the possibility that an OSA and a SB event start contemporaneously should be considered. In practice, however, there are no elements in support of this hypothesis as far as the biological plausibility of such temporal relationship is concerned.

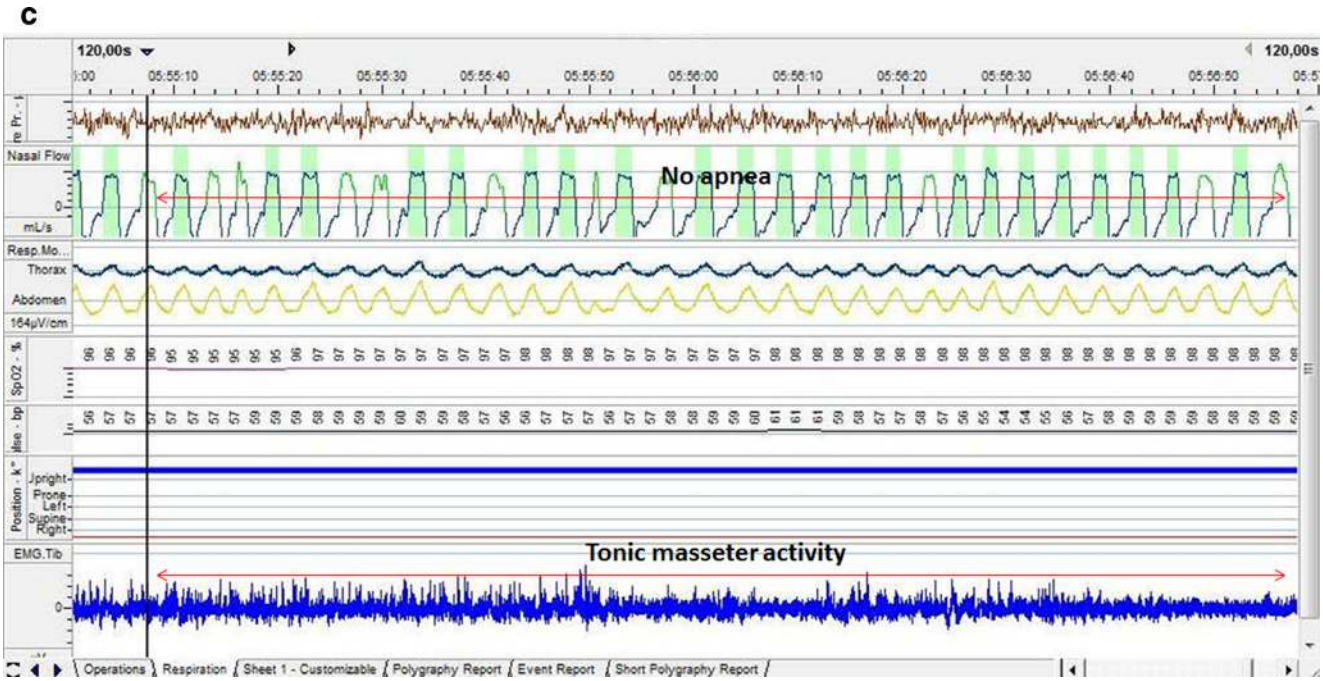


Fig. 2 (continued)

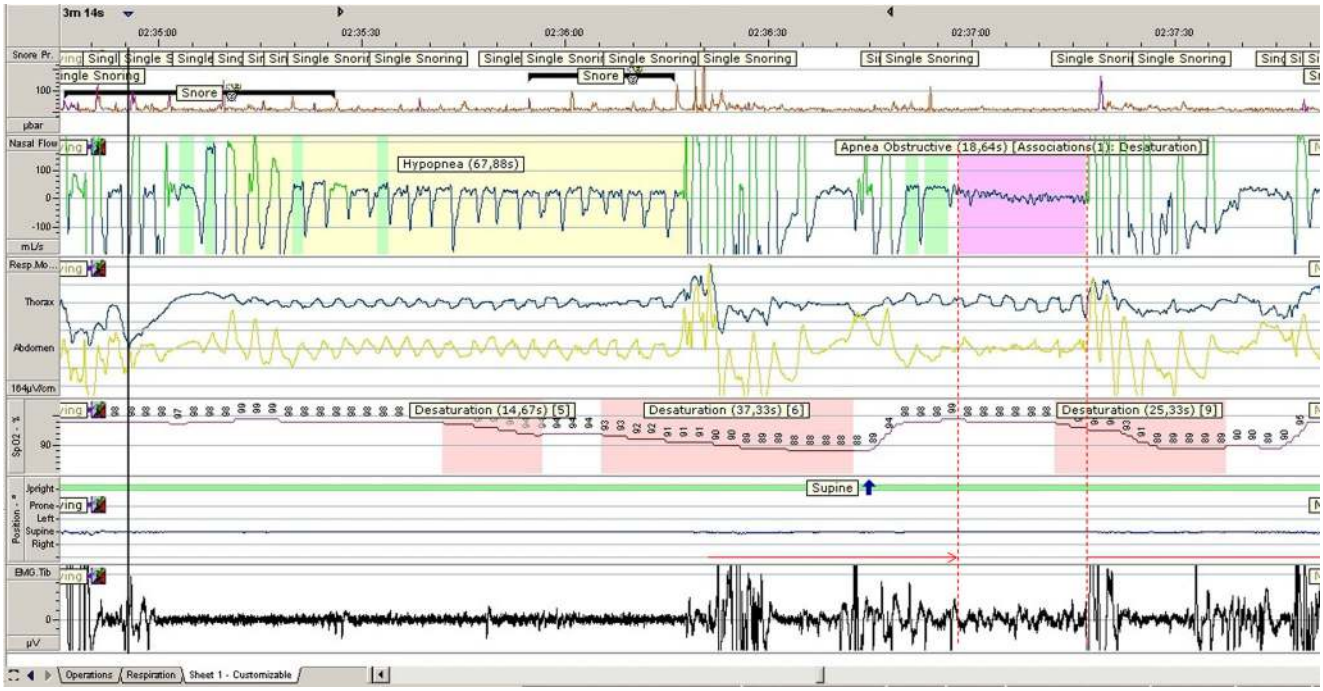


Fig. 3 PSG recording over a 3-min epoch length of a 43-year-old male with moderate OSA (AHI 26.9). Channels are in Fig. 2a. The projection of the OSA event over the masseter EMG trace shows that phasic increases in masseter EMG activity can be shown some seconds before and immediately after the OSA event

Discussion

Literature suggestions that an EMG activity satisfying the features for a SB episode may stop an apneic event stimulated the discussion on the most suitable strategy to assess the temporal SB-OSA relationship. Also, empirical clinical observations that SB reduces after treatment of OSA, regardless of the treatment approach (e.g., continued positive airway pressure (CPAP), oral appliances, mandibular advancement surgery, orthodontic palatal expansion, adenotonsillectomy), seems to support a link between the two phenomena [11, 19, 21, 33, 34].

Notwithstanding that, the available data on the topic is mainly anecdotal, to the point that the association itself between the two phenomena is not evidence-based [14, 35], and the literature is not suitable to answer the fundamental clinical question that underlies their relationship: "Does the activation of jaw muscles follow the apneic episode, thus representing a potential protective mechanism to attempt restoring airway patency by shifting the mandible forward, does it precede the apnea event, is it concurrent with OSA, or is it a temporally unrelated event with respect to the airway obstruction, suggesting that SB and OSA are two distinct sleep disorders?"

In line with what happened with the literature on bruxism and jaw pain, which is similarly inconsistent [5], it is unlikely that any future studies will provide definitive suggestions on the topic until a careful appraisal and evaluation of the different SB and OSA phenomena will be introduced as part of the study designs.

In the present expert opinion, the possible hypotheses for temporal relationships between SB and OSA have been discussed. The most plausible scenario is that the hypothesized temporal relationships between a SB and an OSA event are all actually possible, so that the pathophysiology of the different SB phenomena may vary from a protective role to a causal role with respect to sleep apnea. The relative predominance of one specific sequence of events may vary at the individual level and it can be hypothesized that an OSA-protective role of SB depends, at least in part, on the type and anatomical location of the obstruction. A multidisciplinary approach to the diagnosis of obstruction, involving the assessment of anatomical features, may allow identifying the candidates to present a protective SB and to lend support to the hypothesis that they are likely those OSA patients with an obstruction of the upper airways, viz., at the oropharyngeal level. Based on that, an evaluation of the anatomical location of the airway obstruction site assumes importance as a fundamental criterion to select the patients who should be recruited for future studies on the SB-OSA relationship.

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