Title: Sleep talking vs. sleep moaning: electrophysiological patterns preceding linguistic vocalizations during sleep

Subtitle: Sleep talking and language processing

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ABSTRACT

Sleep talking (ST) has been rarely studied as an isolated phenomenon. Late investigations over the psycholinguistic features of vocal production in ST pointed to coherence with wake language formal features. Therefore, we investigated the EEG correlates of Verbal ST as the overt manifestation of sleep-related language processing, with the hypothesis of shared electrophysiological correlates with wake language production.

From a sample of 155 highly frequent STs, we recorded 13 participants (age range 19-30y, mean age 24.6 ± 3.3; 7F) via vPSG for at least 2-consecutive nights, and a total of 28 nights. We first investigated the sleep macrostructure of STs compared to 13 age and gender matched subjects. We span style="font-family:'Times New Roman'">then compared the EEG signal before 21 Verbal STs vs. 21 Non-verbal STs (moaning, laughing, crying, ...) in 6 STs reporting both vocalization types in Stage 2 NREM sleep.

The 2x2 mixed ANOVA Group x Night interaction showed no statistically significant effect for macrostructural variables, but significant main effects for Group with lower REM (%), TST, TBT SEI and greater NREM (%) for STs compared to controls.

EEG statistical comparisons (paired-samples Student's *t*-Test) showed a decrement in power spectra for Verbal STs vs. Non-verbal STs within the theta and alpha EEG bands, strongly lateralized to the left hemisphere and localized on centro-parietal-occipitals channels. A single left parietal channel (P7) held significance after Bonferroni correction.

Our results suggest shared neural mechanisms between Verbal ST and language processing during wakefulness and a possible functional overlapping with linguistic planning in wakefulness.

Statement of significance

Sleep talking is a non-pathological phenomenon, that has been suggested as possible tool to investigate sleep-related cognitive processing and to directly access sleep mentation. The definition of parasomnias' neural correlates is pivotal for our understanding of sleep-related cognitive processing and their pathophysiological mechanisms. Nonetheless, only few studies defined ST's qualitative features, and no recent evidence allows for a definition of its neural correlate.

We thus carried out a first investigation on ST as direct manifestation of language processing, finding a functional and topographical correspondence with dreamt and planned language production. Our methods allowed to pinpoint the specific EEG correlates of ST, net of the parasomnia-related motor manifestation.

Keywords: Sleep talking, somniloquy, language processing, dream enactment, sleep mentation, parasomnias

INTRODUCTION

Somniloquy, also known as Sleep Talking (ST), is the utterance of speech during sleep, either spontaneous or provoked by external stimuli. ST is distinguished from other generic sleep utterances (laughing, crying, shouting, moaning, sighing),[1] explicitly referring to verbal production, that can vary for length and degree of intelligibility.[2] ST is one of the most widespread parasomnias,[3] with a lifetime prevalence of 66% in the general population.[3] Nonetheless, ST is a benign phenomenon, as attested by its classification within "other symptoms and normal variants" of parasomnias by the International Classification of Sleep Disorders (ICSD-3).[4] The importance of ST is thus only secondary to the concurrent sleep disturbances (i.e., confusional arousal, night terrors, sleepwalking, nightmares, REM Behavior Disorder–RBD, sexsomnia, sleep apnea).

Although a flourishing literature in the early 70s investigated ST quantitative electrophysiological features and correspondence with dream contents[1,2,5–9] only a few recent studies focused on the phenomenon.[10–12] Earlier investigations defined ST as an arousal or stage transition phenomenon, considering it frequently occurs within stage transitions and the second part of the night[4,5,13]; ST was reported to be more freqent in NREM sleep,[2,14] while language productions were more correct and appropriate in REM sleep.[5] Sleep EEG microstructure in the presence of ST was characterized by changes in the theta and alpha activity, accompanied by general motor activation and preceding the speech episode, as predicting the onset of nocturnal vocal activations.[9] Late studies addressed the possibility that motor and vocal activation during sleep could be the actual overt manifestation of the ongoing memory processing, as a formal "overt replay" of recently acquired procedural skills[15] or declarative memories.[11]

Consequently, the interest in parasomnias has been renewed, within the hypothesis of a qualitative and quantitative continuity between nocturnal behaviors and wake cognitive functioning[10–12,15] as well as a possible gateway to sleep mentation.[16–18]

Language during sleep has been mainly studied throughout incoming stimuli.[19–22] Nonetheless, ST implicitly proves that sleep is not incompatible with both overt and covert language production.[17,23,24] Arnulf et al.[10] investigated the formal features of ST language production, finding the decipherable verbal activations to be grammatically correct and consistent with daytime verbal productions. Moreover, vocal activations were respectful of the timing of dialogic exchange, and only a few were self-referred (i.e., in the first person, as one would expect in a reflexive context, also known as "inner speech"[25]). This finding endorsed the perspective that sleep-spoken words could be a direct manifestation of the patients' dreaming mind, specifically of a dreamed linguistic interaction.[10] Consistently, the neurobiology of vocal production in sleep may identify patterns overlapping verbal production in wakefulness. Investigations on dream speech (i.e., the presence of dialogic elements within the dream reports collected upon awakening) propose an overlapping between the neural circuits underlying linguistic production in dreams and those known in waking life.[17,26–28] Phasic electromyographic activations appear to be present in the speech muscles also during dream speech,[27] while the activation of the muscles involved in language production is a prerequisite for the appearance of verbal productions.

The idea of a systematic investigation of somniloquy is intriguing for understanding a plethora of cognitive processes in sleep: the activation (or replay, whether cognitive or neural) of memories, autobiographical or episodic; the perception of auditory (e.g., words spoken by an imaginary interlocutor) and/or visual (the dream scene) stimuli in the absence of an external percept (i.e., incoming stimuli); the preparation for a

linguistic response, resulting in a motor-linguistic output, that may be contained exclusively within the dream scene (dream speech) or become manifest (sleep speech).

In our research, we aimed at investigating ST as an isolated phenomenon and verbal vocal activation as evidence of an electrophysiological and topographical specificity of linguistic production in sleep, i.e., as a "direct observation" of the processes of linguistic elaboration. Coherently, we carried out the first original investigation on the electrophysiological scenario immediately preceding the sleep language production.

The hypothesis was that the presence of characteristic EEG activity predictive of vocal activation (Verbal ST) could be peculiar to the verbal phenomenon itself and indicate an ongoing sleep-related language processing, distinguishing the linguistic activations from other non-verbal utterances (Non-verbal ST) in its neurobiological correlates.

METHODS

Participants

We carried out a three-phases selection procedure (Figure S1), including an online survey to rule out other sleep or psychiatric disorders and an at-home monitoring to objectively verify presence and frequency of the phenomenon prior in laboratory experimental recording (see Supplemental materials for a detailed description of the survey and participants' selection criteria). Within a total of 1309 questionnaires, N = 155 (age range 18-69y, mean age 26.32 ± 7.84; F 107) participants declared themselves as Highly frequent STs. From this sample, we selected N = 50 Highly frequent STs for home monitoring. During the home monitoring week, the participants produced 766 vocalizations (385 Non-verbal STs and 381 Verbal STs, 213 of which non-intelligible, for a total of 607 and 20414 intelligible characters) and 13 subjects (age range 19-30y, mean age 24.6 ± 3.3, 7F; 1 left-handed, 2 ambidextrous) were suitable for EEG recordings.

The participants signed informed consent before participation in the study. The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the Department of Psychology of the Sapienza University of Rome (protocol number: 0001288).

Procedures

We tested the 13 participants for at least two consecutive nights of undisturbed sleep: an adaptation night, to avoid a possible first night effect on the macro- and microstructural characteristics of sleep[29]; one or more experimental nights, during which we pursued the conditions of Verbal ST (one or more episodes of ST with

verbal vocalizations, whether intelligible or not) and Non-verbal ST (any grumbling, moaning, laughing, crying, long sigh).

The experimental sessions started at 7 p.m. After completing the EEG headset assembly, the participants were placed in an acoustically isolated, temperature-controlled, audio/video-monitored bedroom for the vPSG recording.

We guaranteed at least 8 hours of bedtime. The final awakenings were carried out at the end of the experimental night and necessarily after about 7 hours of undisturbed sleep. Upon the final awakening, the participants filled out the sleep- and dream log.

EEG recordings

We acquired the PSG signal through a 32-channel system (BrainAmp MR Plus System, Brain Products GmbH, Gilching, Munich) and the dedicated BrainVision Recorder software for online visualization. The data relative to the cortical activity of the subjects (EEG) were acquired from the scalp through 28 cortical derivations (Fp1, F3, F7, Fc1, Fp2, F4, F8, Fc2, Fc5, C3, T7, Cp1, Fc6, C4, T8, Cp2, Cp5, P3, P7, O1, Cp6, P4, P8, O2, Fz, Cz, Pz, Oz), positioned according to the international 10-20 system, with reference on averaged mastoids (A1, A2), and ocular and muscular activity were recorded by EMG and EOG electrodes. The impedance of the electrodes was kept below 5 kOhms.

PSG signals were recorded at a sampling rate of 250 Hz. EEG signals were high pass filtered with time constant of 1-s and low pass filtered at 70 Hz. Possible interference from the electricity network was filtered by a notch filter at 50 Hz (± 5Hz). The filters were implemented as phase shift-free Butterworth filters.

Data analysis

Presence and frequency of ST in the general population and incidence of other altered nocturnal behaviors (online survey) in ST subjects. We reported the presence and frequency of altered nocturnal behaviors as total numbers and percentages for each MUPS item/behavior. The presence and frequency of the single behavior was reported for the Highly frequent ST group only, separately for lifetime frequency (i.e., participants who answered 2 “Was observed years ago but not anymore” for the single behavior) and current frequency (i.e., participants who reported a score ranging from 3 “Very seldom–less than once per year” to 7 “Very frequently–every or almost every night”).

Sleep macrostructure. The 13 suitable Highly frequent ST participants were recorded for a total of 28 nights (15 experimental nights). EEG scoring has been carried out for 20-s epochs, according to Rechtschaffen and

Kales' criteria.[30] We then identified the sleep stage corresponding to each Verbal and Non-verbal STs. We then computed the following sleep parameters: sleep stages latencies, duration (in minutes) and percentages; total sleep time (TST), total bedtime (TBT), sleep efficiency index (SEI), number of awakenings, number of arousals, intra-sleep wakefulness (ISW), ST frequency over total sleep time (ST number/TST).

To investigate the sleep structure in our Highly frequent STs and to assess the hypothesis of a more fragmented and lighter sleep in STs, the macrostructural variables of the two consecutive nights were additionally compared to an archive dataset of sleep recordings in healthy controls. The selected dataset included 13 age- and gender-matched healthy controls (age range 18-28y, mean age 23.62 ± 2.96; 7F).

EEG quantitative analysis. The following analyses were performed for participants (N = 6) who produced the combination of Verbal ST (intelligible and non-intelligible) and Non-verbal ST (mumbling, laughter, moaning, crying, long sighs) during the experimental nights. The analysis has been performed for stage 2 NREM sleep only, as the only sleep stage in which we obtained an adequate sample size to perform subsequent statistics.

To verify that the electrophysiology of the sleep period preceding the analyzed phenomenon is peculiar and indicative of the verbal phenomenon itself, the EEG analysis has been then carried out on the 20-s interval preceding each Verbal and Non-verbal STs. The sleep interval was chosen relying on the available literature investigating motor activations prior to sleepwalking.[31] Each sleep interval was free of any other motor behavior (i.e., no other parasomnias-like motor activation was present, except for Verbal or Non-verbal STs). The rationale was that this comparison would clarify the specific EEG correlate of verbal vocal production, net of the motor activation necessary for generic (verbal and non-verbal) vocalizations. Moreover, to verify the time-locking of the selected STs to the chosen sleep interval within the N = 6 participants, we selected an equivalent sleep period that preceded each Verbal and Non-verbal STs by 2 minutes (Baseline), with the rationale that a substantial difference between the electrophysiological pattern observed in the comparison between Verbal vs. Non-verbal STs would confirm the goodness of the observed phenomenon. To allow for this further analysis, Verbal ST and Non-verbal ST sleep interval were pooled together (i.e., Vocalizations) and subsequently compared to the corresponding Baseline. We then sampled the selected sleep interval and paired them by sleep cycle.

To further allow for artifact rejection due to muscle activations or eye movements, the last 20 seconds before each Verbal and Non-verbal ST and Baseline have been sectioned in 4-second epochs. The data obtained for the 28 cortical derivations were offline passband filtered between 0.33 Hz and 30 Hz (second order Bessel filter) and then underwent spectral analysis using FFT (Fast Fourier Transform) at 0.25 Hz frequency

resolution. The spectral power for the canonical EEG bands [delta (0.5-4.75 Hz), theta (5.00-7.75 Hz), alpha (8.00-11.75 Hz), sigma (12.00-15.75 Hz) and beta (16.00-24.75 Hz)] was obtained by averaging across adjacent bins and subsequently log transformed.

Statistical analysis

Sleep macrostructure in ST subjects. To investigate the hypothesis of a fragmented and less efficient sleep in Highly frequent STs, the macrostructural variables of the two consecutive nights were compared to sleep recordings of healthy non-parasomniac subjects. To this aim, two-way mixed design ANOVAs Group (STs vs. Controls) x Night (Adaptation vs. Experimental night) were carried out on PSG variables. Bonferroni correction for multiple comparisons was computed to adjust the probability value to reject the null hypothesis. Considering the macrostructural variables are mutually correlated variables, an intercorrelation index between the macrostructural variables has been calculated[32,33] ($r = 0.0037$). Considering this correlation and the number of statistical comparisons made (48), the value of critical α was thus adjusted to ≤ 0.0014.

EEG patterns preceding linguistic vocalizations

Power spectra of ST episodes for each EEG frequency band were compared (ST Verbal vs. Non-verbal ST; Vocalizations vs. Baseline) by means of paired samples Student's *t*-tests, separately for each derivation, in stage 2 NREM sleep. Bonferroni correction for multiple comparisons was computed to adjust the probability value to reject the null hypothesis. Considering that EEG data are mutually correlated variables, an intercorrelation index between dependent variables has been calculated[32,33] separately for the comparison Verbal ST vs. Non-verbal ST in Stage 2 NREM ($r = 0.5047$) and the comparison Vocalizations vs. Baseline ($r = 0.5404$), for the last 20 seconds of sleep. Considering these correlations and the number of statistical comparisons made (140), the value of critical α was thus adjusted: ≤ 0.0043 ($t \geq -4.48$) for ST Verbal vs. Non-verbal ST comparisons in Stage 2 NREM; ≤ 0.0051 ($t \geq 2.96$) for Vocalizations vs. Baseline comparisons in Stage 2 NREM.

Results

Presence and frequency of ST in the general population and incidence of other altered nocturnal behaviors in ST subjects (online survey). Within the 1309 participants to the online survey (age range 18-70y, F = 894–68.35%), 71.96% (N = 942) declared to be ST or that they talked during sleep over years. 11.8% (N = 155,

age range 18-69y, mean age 26.32 ± 7.84; F = 107–69.03%), presented ST weekly or daily, and have thus been considered Highly frequent STs.

Figure 1 shows the percentages and number of participants among the Highly frequent STs (N=155) reporting a presence of other altered nocturnal behaviors from 3 “Very rarely–less than once a year” to 7 “Very often–every or almost every night”. The percentages and number of participants who experienced other altered nocturnal behaviors in the past but not currently (“It was observed years ago but is no longer present”) are reported in Figure S2.

The nocturnal behaviors more often related to ST in the literature show the highest percentages in co-presence (such as arousal disturbances in falling asleep, NREM parasomnia-related phenomena and violent or dream enactment behaviors).

[Please, insert Figure 1 about here]

Sleep macrostructure in ST subjects. Within the Highly frequent STs sample, N = 2-night recordings were excluded from the total of the nights under examination (N = 28): N = 1 because of technical issues during the experimental sessions that did not allow the completion of the polysomnographic session, and N = 1 because occurred at distance from the two nights considered, therefore it was not considered an adaptation night. Descriptive statistics for the macrostructural variables in the adaptation night and the experimental night of the 13 ST participants and the 13 matched healthy controls are reported in Table 1.

[Please, insert Table 1 about here]

Table 2 reports the results for the 2x2 mixed ANOVAs (Group x Night). The Group x Night interaction showed no statistically significant effect after Bonferroni correction. For the main effects, no statistical significance was found for Nights, while we found significant effects for Group. STs compared to controls had lower REM (%) (F = 30.2; p = <.0.001; η2p = 0.557) and higher NREM (%) (F = 14.2; p = <.001; η2p = 0.372). TST (F = 55.7; p = <.001; η2p = 0.699), TBT (F = 42.2; p = <.001; η2p = 0.637) and Sleep efficiency index (F = 15.0; p = <.001; η2p = 0.384) showed lower mean values for STs compared to controls.

[Please, insert Table 2 about here]

Vocal activations obtained in the laboratory setting. The participants produced 165 STs (97 Non-verbal STs, 35 non-intelligible Verbal STs, 33 intelligible Verbal STs) during 21 of the 28 nights. After offline scoring, 10 Non-verbal STs were scored as Wake or Stage 1, 69 in Stage 2, 9 in SWS, 9 in REM stage STs; as far as Verbal STs are concerned, no difference was made for the final selection between intelligible and non-intelligible verbal activations. Of these, 1 were in Wake, 51 were in Stage 2, 12 in SWS, 4 in REM stage. STs offline scored as Wake or Stage 1 vocalizations were excluded from the subsequent analysis. Consequently, the indexes reported in Table 3 refer to statistical comparisons (paired-samples Student's *t*-Tests) for Night 2 vs. Night 1, for a total of 26 PSG recordings (13 experimental nights vs. 13 adaptation nights, occurred in two consecutive night sessions), and a total of 154 vocalizations. No significant differences were found for vocal activations between the two nights.

[Please, insert Table 3 about here]

EEG specificity of the sleep interval considered in relation to ST. Figure 2 and Table S1 show the descriptive statistics for the topographic distribution of EEG power values, divided by canonical EEG bands, for the two conditions Vocalizations and Baseline, and the results of the statistical comparisons between them by paired sample Student's *t*-tests. Increased EEG activity was observed in the delta, theta, alpha and beta bands for the Vocalizations group compared to Baselines, while no difference was significant for the sigma band between the two groups. Following Bonferroni's correction, the difference in the delta band in correspondence of frontal, central and temporal derivations appeared statistically robust. The same pattern, albeit more pronounced on the frontal leads, was observed for the theta frequency band. The alpha frequency band maintained a generalized significance almost to the entire scalp. Finally, while no significance was observed for the sigma frequency band, two specific derivations (C4, Fc2) were significant for the beta band. In conclusion, a generalized increase was observed especially for the slow (delta and theta) and medium frequency (alpha) bands, which seemed peculiar to the vocalization onset. This result strengthens the results of the main comparison analyses between Verbal STs vs. Non-verbal STs in Stage 2 NREM, and the spectral powers for the theta and alpha band seemed to be characteristic and predictive of the verbal vocal activation onset, and time-locked to the sleep interval considered.

[Please, insert Figure 2 about here]

Electrophysiological specificity for Verbal STs vs. Non-verbal STs. The reported statistics refer to the comparisons between 21 Verbal STs and 21 Non-verbal STs within Stage 2 NREM in N = 6 Highly frequent STs (age range 19–27y, average 23.83 ± 3.6; 3 F; 6 right-handed). Figure 3 shows the topographic distribution for the mean spectral powers in the canonical EEG bands for Verbal and Non-Verbal ST and the results of the statistical comparisons.

[Please, insert Figure 3 about here]

The statistical comparisons (paired-samples Student's *t*-Test) for the 20-second sleep interval preceding each vocalization within the two conditions (21 ST Verbal STs vs. 21 Non-verbal STs) showed a selective decrement in EEG power for the theta and alpha band as characteristic and predictive of the verbal voice activation onset in stage 2 NREM. Following Bonferroni's correction for multiple comparisons, the difference appeared statistically robust in correspondence of a single left parietal derivation for the theta band (P7, t = - 4.48, p = 0.0002), and for the alpha band (P7, t = - 3.29, p = 0.0037).

DISCUSSION

Our results point to continuity between the cortical activity involved in linguistic production during wakefulness and verbal production during sleep. Specifically, the selective decrease for the theta and alpha bands over the left temporo-parietal-occipital derivations for the Verbal STs suggests an ongoing elaboration of language in sleep. These correlates are consistent with what is expected from a purely linguistic phenomenon,[34,35] and with studies on the topography of linguistic planning and production during wakefulness.[36–39] A study investigating covert and overt word generation found a specific decrease in the fronto-temporal theta activity as a predictive correlate of language production.[36] Additionally, the reduction in the theta and alpha power is consistent with the available literature about dream speech, highlighting a decrease in these frequency bands, strongly lateralized and focused on the left front-temporal-parietal regions.[26,28] Hong et al.[26] found a decrement in EEG power for the alpha band focused on Broca's (C3) and Wernicke's (P3) language areas, proportional to the amount of expressive and receptive language reported in dreams[26,27]; Noreika et al.[40] investigated the EEG correlates of hypnagogic hallucination contents in a single-case study, reporting a specific decrease in EEG powers for the theta and alpha bands for episodes of linguistic hypnagogic hallucination.[40] This overlap suggests that the cognitive processes in place before the overt vocal production could be related to sleep mentation, containing a specific language interaction, thus giving promising insight on the possible implication of our results. However, most of the literature on

language production in wakefulness points to high-frequency EEG power, such as gamma and beta, as an EEG correlate of verbal production in wakefulness,[37,41–44] coherently with current evidence of an actual increment in high-frequency activity on the left posterior temporal regions (Wernicke's area) when dreams containing linguistic production were reported.[28]

Importantly, the investigation over the sleep periods preceding the onset of verbal vocalization, compared to non-verbal vocalization, is quite original in the literature. For this reason, there are currently no evidence in the literature for an adequate comparison. Thus, additional investigations are needed to pinpoint the actual neural dynamics of covert language production and language processing in sleep.

Thanks to our control condition (i.e., the Vocalizations vs. Baseline comparison) (see Figure 2 and Table S1), we could provide an additional proof of concept regarding the time-locking of our principal results. Coherently, the increase of EEG activity for the Vocalizations condition should be specific to the parasomnias' motor manifestation, thus remarking the temporal and functional association of the examined EEG pattern to the verbal nature of ST. The current literature about parasomnias could not pinpoint the specific pathophysiological sleep mechanism allowing the behavioral manifestation. Relying on RBD theoretical literature, the behavioral manifestations in sleep could be a direct indication of cortical activation[45–47] or a mere manifestation of reflex and innate behavior.[48,49] Specifically, Iranzo[50] suggested that the qualitative aspects of RBD vocalizations could derive by the involvement of different patterns of brain activation in sleep, which reflect the processes of linguistic production in wakefulness. Moreover, Iranzo[50] posited that a) the abnormal activation of a first circuit is responsible for the voluntary initiation of vocal behavior, specifically innate vocal reactions such as emotional non-verbal vocalizations (crying, laughter, shouting); b) the abnormal activation of a second circuit is responsible for the production of learned (complex) vocal patterns.[50] Our results are coherent with the hypothesis of ST as a direct prouct of the activation of superior cortical structures, therefore a primary manifestation of the ongoing cognitive processing. This statement gains values thanks to the comparisons between Verbal and Non-verbal STs, which rules out the alternate hypothesis of an observation of the parasomnia's motor disinhibition.

To assess whether somniloquy affects the macrostructural characteristics of the sleep in the direction of higher fragmentation, we performed a comparison with an archive dataset of healthy controls. According to the hypothesis, STs had lower sleep efficiency than controls. However, number of arousals, awakenings, and intra-sleep wakefulness, although coherent with the hypothesis of higher sleep fragmentation in STs, did not reach statistical significance after correcting for multiple comparisons. Interestingly, a different between-groups pattern was observed for NREM and REM sleep, with lower REM and higher NREM in STs. These

results are indeed coherent with the findings of a general presence of vocalizations in NREM stages for our samples, although we have no direct explanation for this increased amount of NREM sleep.

Lastly, although our research did not aim at investigating the prevalence of ST in the general population, our online survey confirmed its substantial prevalence (71.96%, N = 942). Within this sample, 11.8% of subjects (N = 155) declared themselves as frequent ST. It is important to point out that the questionnaires used to verify sleep quality and the presence and frequency of the phenomenon are not strictly focused on the ST itself, but rather generic questionnaires of investigation on altered sleep behavior and sleep quality. The survey administered[51] verified presence and frequency of altered nocturnal behaviors but included a single item of reference to ST ("Talking during sleep") and one item referring to possible non-verbal vocalizations with implicit negative connotation ("Loud and repeated sighing and moaning during sleep"), not allowing to specifically distinguish emotionally neutral vocalizations or the quality of verbal production. Ad hoc questionnaires should thus be developed, also involving the participants' bedpartners or roommates, to investigate specific features of ST. Nevertheless, our procedures exceed these limitations. Thanks to the at home-monitoring we further verified basic features and frequency of the phenomenon for fifty Highly frequent STs. We recorded a total of 766 voice activations, although most of them were non-verbal or unintelligible. Further investigations on the formal characteristics of vocal productions are needed, in order to enrich the available findings about the continuity between night and daytime vocal productions, and of a "dreamed" dialogical exchange (see Arnulf et al.[10]).

LIMITATIONS

The aim of defining the capacity of ST to access cognitive elaboration in sleep, even if partially achieved, cannot be considered completely definitory, especially because of the model used. As a matter of fact, our intention to investigate the phenomenon in its predictive EEG characteristics clashed with the limit of a small number of conditions obtained in SWS and REM. The higher observation of STs in Stage 2 NREM could be due to a higher percentage of this stage of sleep in both the adaptation and experimental nights. Moreover, we observed a higher number of vocal activations during the second night, corresponding to a substantial increase in the duration and percentage of NREM sleep, partly due to the higher presence of Stage 2 NREM. Nonetheless, no significant difference between the two nights was found when calculating the frequency rate of ST in nights 1 and 2, in relation to the TST index. It could therefore be hypothesized that the process of selection of the experimental subjects (i.e., the choice to investigate the phenomenon within a healthy sample, in the absence of other associated parasomnias) has led to a lessening in the observation of ST in REM and SWS, known to be mostly related to purely pathophysiological phenomena

(i.e., the complex motor activities in sleepwalking, or the violent motor and vocal activations during the increased electromyographic activity in RBD). ST could be thus confirmed as a beneolent phenomenon net of other parasomnias, not easily observable in the absence of other pathophysiological mechanisms. However, the lack of a specific literature on the electrophysiology of ST under clinical conditions makes this statement speculative. Moreover, thanks to the laboratory control over the conditions, we objectively verified the number and quality of the vocal activations produced, while other forms of artifact could have affected our results. The setting of the laboratory is in fact not ecological, thus being able to interfere with the natural manifestation of the phenomenon, as much as already known for the macro- and microstructural parameters of normal sleep[29] and for the qualitative aspects of dream recall and dream production.[52] The choice to record undisturbed sleep nights was dictated by the need to not interfere with the quality of sleep of the experimental subjects, considering the lack of evidence about the quality of sleep of these subjects. This choice, however, did not allow an effective observation of all the possible features of mental processing and ST; nonetheless, the experimental interruption of sleep could stimulate sleep speech, as proven by the literature on sleep deprivation and precipitating factors for other parasomnias to which it is associated [53], and thus being desirable in future studies.

CONCLUSIONS

We reported the first systematic investigation of the electrophysiological correlates of somniloquy, with the aim of investigating its involvement in cognitive processing in sleep, with a particular focus on sleep-dependent language processing. In conclusion, the results support the view of sleep talking as overt manifestation of sleep-related language processing and legitimize the potential use of parasomnias as a model for direct observation of sleep-dependent cognitive processing.

As a matter of fact, the observation that at an interval of time prior to the onset of the phenomenon corresponds to a purely linguistic output, suggests that the phenomena in action before verbal production are of a linguistic programming and elaboration. Anyway, this is only a speculative hypothesis and only a direct study comparing the EEG signal prior to sleep vocalizations to similar conditions at wake would identify if our pattern of EEG differences is specific of sleep talking or it points to shared mechanisms between sleep and wake.

Concluding, we suggest these methods could be generalized to sleep related behavioral manifestations, and the study of electrophysiological predictability of parasomnias could be elected as gold standard for the study of cognitive processing in sleep, specifically focusing on the distinction of the motor manifestation by its cognitive correlates.

AUTHORS CONTRIBUTION

Conceptual: AM, AD, SS, MC, VA, LA, MG, MP, LDG. Investigation: AM, AD, SS, VA, LA, LDG. Data Curation: AM, AD, MC, VA. Writing: AM, AD, SS, LDG

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FINANCIAL DISCLOSURE

None.

NON-FINANCIAL DISCLOSURE

None.

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FIGURES

Figure 1. Current prevalence of ST.

Other altered nocturnal behaviors (MUPS frequency from 3 "Very seldom - less than once a year" to 7 "Very frequently - Every or almost every night") currently experienced by Highly frequent STs (N=155).

**The single items do not refer to a diagnosis but to a qualitative description of the phenomenon.*

Figure 2. EEG specificity of the sleep interval considered in relation to ST.

[Top and central rows] Topographic distribution of spectral powers for the 20-second sleep interval in Stage 2 NREM for each frequency band, delta (0.5 - 4.75 Hz), theta (5.00 - 7.75 Hz), alpha (8.00 - 11.75 Hz), sigma (12.00 - 15.75 Hz) and beta (16. 00 - 24.75 Hz), preceding Verbal and Non-verbal STs (Vocalizations, top row) and the 20-second sleep interval of the 2 minutes preceding each voice activation, as a baseline (Baseline, bottom row). The red colour range indicates higher EEG power values, the blue colour range indicates lower power values.

[Bottom row] Statistical comparisons (paired-sample Student's t-test) of the last 20 seconds preceding the Vocalizations condition (Verbal and Non-verbal vocal activations) vs. Baseline (2 minutes preceding each vocalization) in Stage 2 NREM for each frequency band delta (0.5-4.75 Hz), theta (5.00-7.75 Hz), alpha (8.00-11.75 Hz), sigma (12.00-15.75 Hz), and beta (16.00-24.75 Hz). The red colour range indicates an increase in spectral powers in the Vocalizations condition. The derivations in which the differences are statistically significant following Bonferroni correction are highlighted in white ($t \geq 2.96$, $p = \leq 0.0051$).

Figure 3. Electrophysiological specificity for Verbal STs vs. Non-verbal STs.

[Top and central rows] Topographic distribution of the power spectra of the 20-second sleep interval in Stage 2 NREM for each frequency band: delta (0.5-4.75 Hz), theta (5.00-7.75 Hz), alpha (8.00-11. 75 Hz), sigma (12.00-15.75 Hz) and beta (16.00-24.75 Hz) of the 20-second interval of sleep in Stage 2 NREM preceding verbal vocal activations (Verbal STs) vs. non-verbal vocal activations (Non-verbal STs). Colours in the range of red correspond to higher EEG power values, colours in the range of blue indicate lower power values.

[Bottom row] Statistical comparisons (paired-sample Student's t-test) of the last 20 seconds preceding the Verbal ST vs. Non-verbal ST conditions in Stage 2 NREM for each frequency: band delta (0.5-4.75 Hz), theta (5.00-7.75 Hz), alpha (8.00-11.75 Hz), sigma (12.00-15.75 Hz) and beta (16.00-24.75 Hz). The blue colour range indicates a decrease in spectral powers in the Verbal ST condition. The derivation in which the differences are statistically significant following Bonferroni correction ($p < 0.0043$), highlighted in white, is P7 for theta ($t = -4.48$, $p = 0.0002$) and alpha ($t = -3.29$, $p = 0.0037$) frequency bands.

TABLES

Table 1. Sleep macrostructure in sleep talkers: descriptive statistics.
Descriptive statistics for the macrostructural variables in the adaptation night (Night 1) and the experimental night (Night 2) for N = 13 Highly frequent STs and N =13 Healthy controls.

Abbreviations: ISW = intra-sleep wakefulness; TBT = total bedtime; #MA = number of movement arousal; TST = total sleep time; %SEI = sleep efficiency index.

Table 2. **Sleep macrostructure in ST subjects: statistical comparisons with healthy controls.**
2x2 mixed ANOVAs Group (STs vs. Healthy controls) x Night (Adaptation vs. Experimental night) for the macrostructural variables. The table shows Fisher's F coefficients (F), the associated probability (p), and the effect size (partial eta-squared, ηp2). Significant results after Bonferroni correction ($p = 0.00104$) are highlighted in bold.

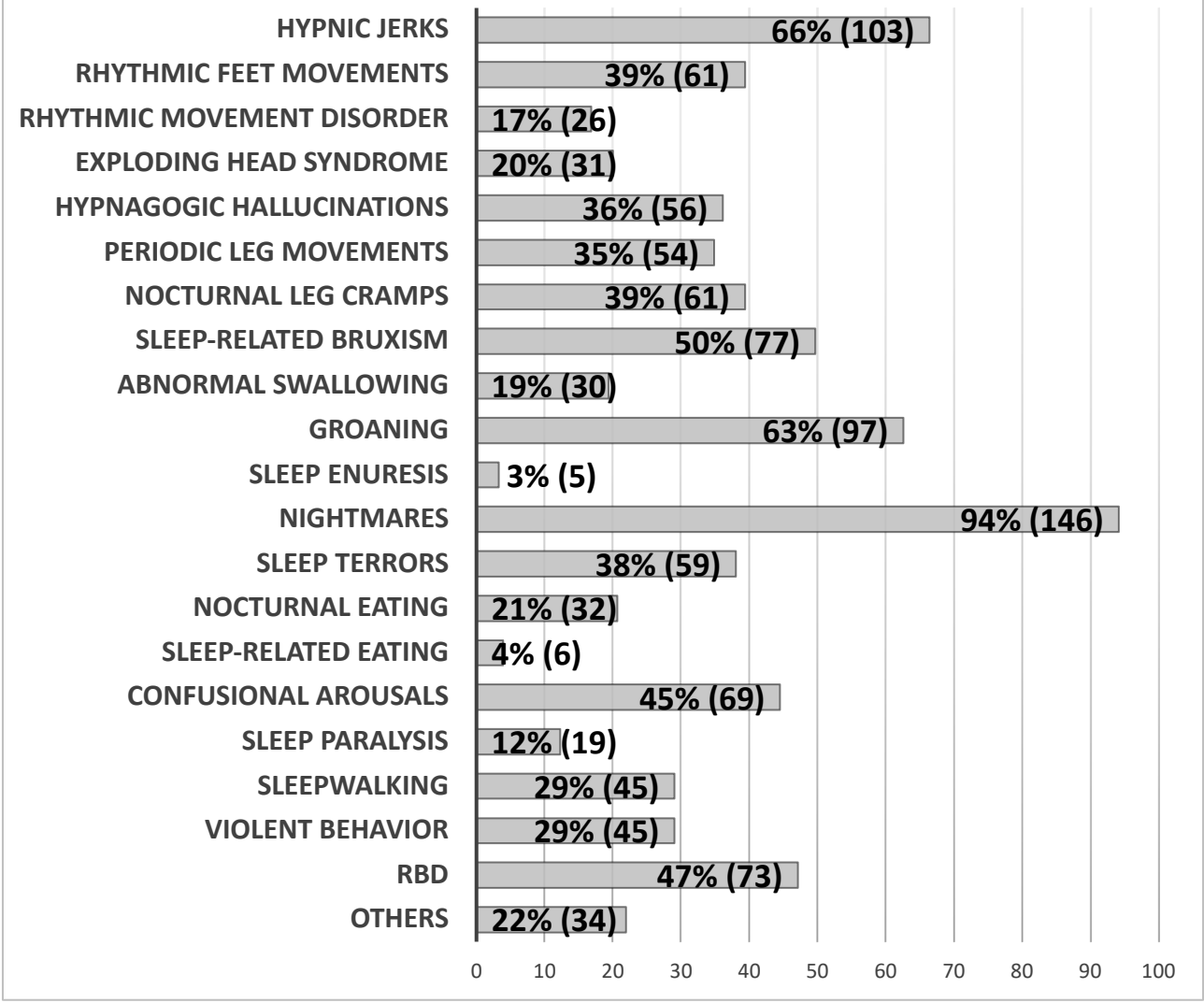
Abbreviations: ISW = intra-sleep wakefulness; TBT = total bedtime; #MA = movement arousal number; TST = total sleep time; %SEI = sleep efficiency index.

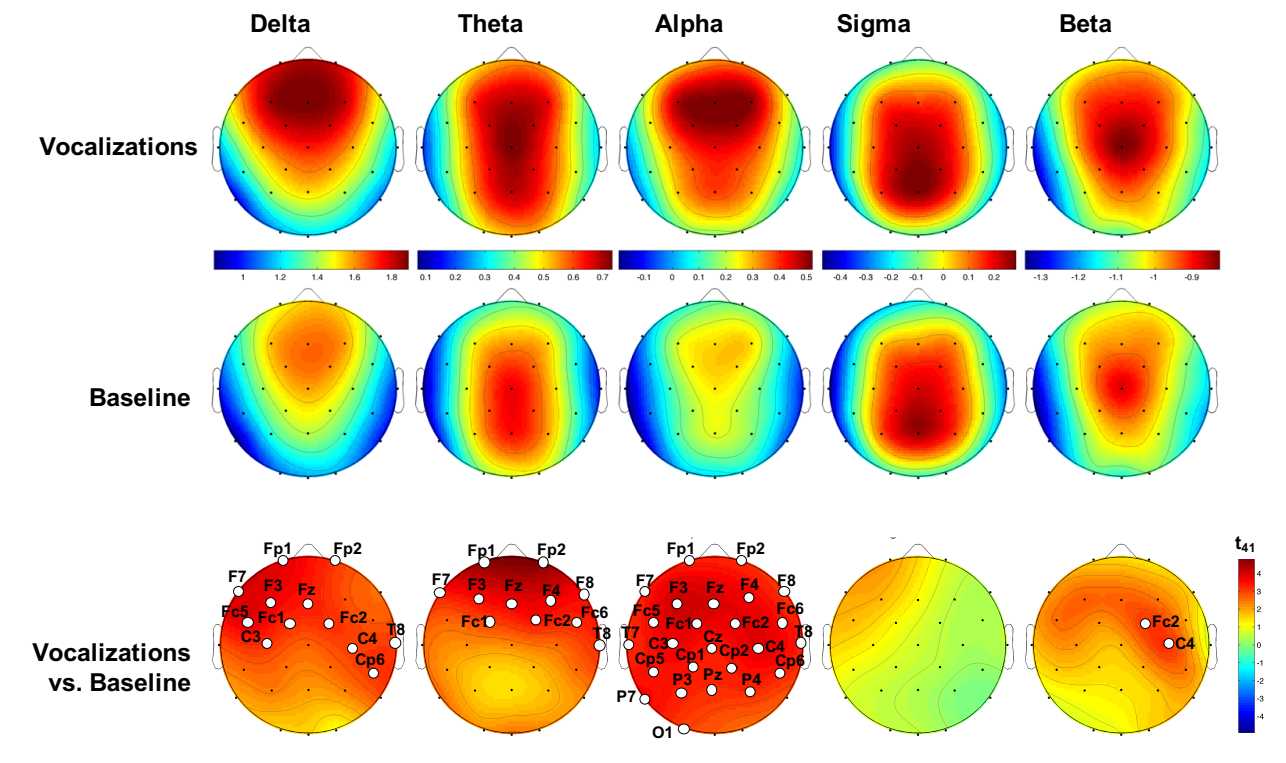
Table 3. Vocal activations obtained in the laboratory setting.
Statistical comparisons (paired samples *t*-Tests) for the Sleep talking episodes registered during the experimental night (Night 2) and the adaptation night (Night 1) for N = 13 participants. The first column shows the raw number of voice activations, the ratio of voice activations to total sleep time; the second and third columns show mean and standard deviation for Night 1 and Night 2; the fourth column shows *t*-values, and the last column shows the associated probability (p values).

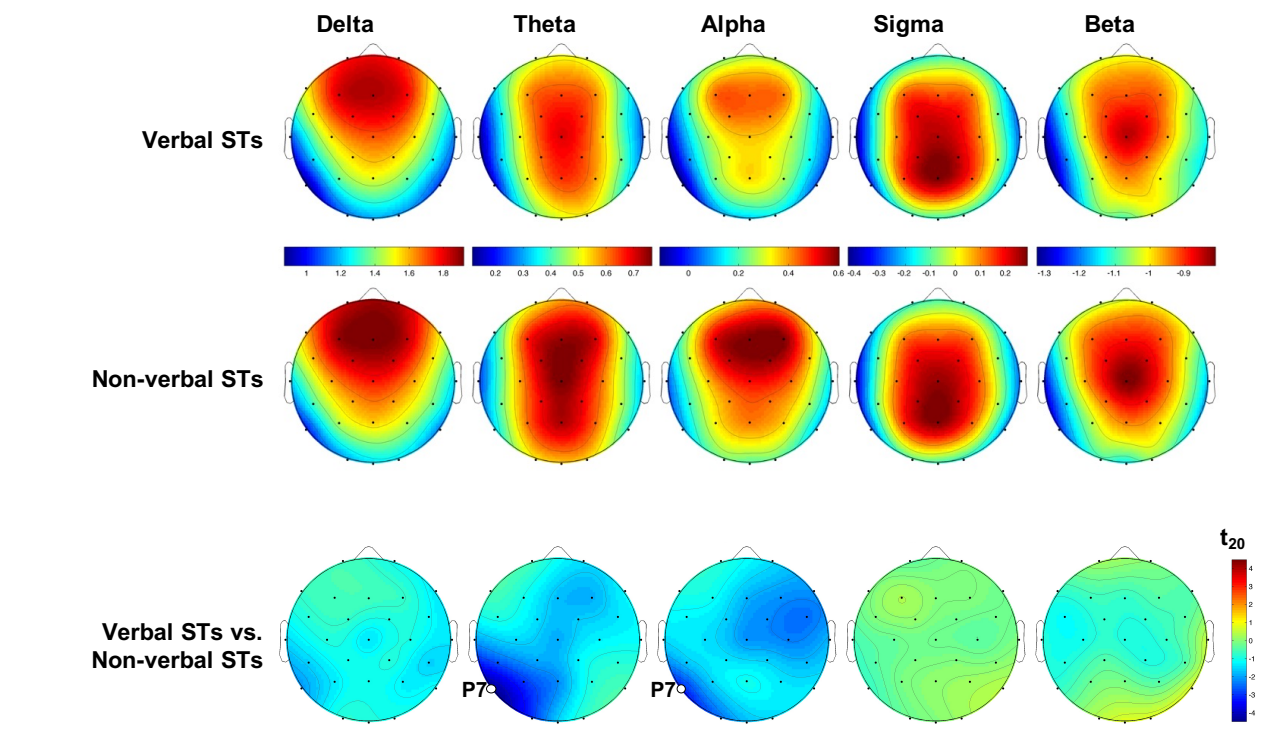
Abbreviations: ST frequency = ST frequency over total sleep time (ST number/Total Sleep Time).

1

Current prevalence of other altered nocturnal behaviors in Highly frequent STs*




Delta
Theta
Alpha
Sigma
Beta
Vocalizations
1 1.2 1.4 1.6 1.8
0.1 0.2 0.3 0.4 0.5 0.6 0.7
-0.1 0 0.1 0.2 0.3 0.4 0.5
-0.4 -0.3 -0.2 -0.1 0 0.1 0.2
-1.3 -1.2 -1.1 -1 -0.9
Baseline
Vocalizations vs. Baseline
Fp1 Fp2 F7 F3 Fz F4 F8 Fc5 Fc1 Fc2 Fc6 C3 C4 T7 T8 Cz Cp5 Cp1 Cp2 Cp6 P7 P3 Pz P4 O1
Fc2 C4
t41
4 3 2 1 0 -1 -2 -3 -4


Delta
Theta
Alpha
Sigma
Beta
Verbal STs
1 1.2 1.4 1.6 1.8
0.2 0.3 0.4 0.5 0.6 0.7
0 0.2 0.4 0.6
-0.4 -0.3 -0.2 -0.1 0 0.1 0.2
-1.3 -1.2 -1.1 -1 -0.9
Non-verbal STs
Verbal STs vs.
Non-verbal STs
P7
P7
t20
4 3 2 1 0 -1 -2 -3 -4