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ORIGINAL RESEARCH

Comparison of EMG power during sleep from the submental and frontalis muscles

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Objective: Submentalis electromyography (*s*EMG) and frontalis electromyography (*f*EMG) muscle activities have been used to assist in the staging of sleep and detection of disruptions in sleep. This study was designed to assess the concordance between *s*EMG and *f*EMG power, by and across sleep stages.

Methods: Forty-three records with simultaneous acquisition of differential signals from the submental and frontalis muscles were evaluated. Sleep stages were assigned using the polysomnography signals based on majority agreement of five technicians. The *s*EMG and *f*EMG signals were identically filtered and aligned prior to cross-correlation analysis.

Results: A strong concordance between *s*EMG and *f*EMG power was observed, with 95% of the records exhibiting at least moderate agreement. During rapid eye movement (REM) sleep, *s*EMG power was significantly less than *f*EMG power, but exhibited four times greater across-subject variability. *f*EMG power during wake and non-REM (NREM) sleep was greater than *s*EMG power, but with 50% less variability. Differences in wake and N1 mean power and between the other sleep stages were more distinct in the *f*EMG recordings. Relative changes in *s*EMG and *f*EMG power across wake, NREM, and REM stages were essentially identical with median by-subject cross correlations of 0.98 and interquartile ranges of 0.97 and 0.99, respectively.

Conclusion: The *f*EMG and *s*EMG power values were similar during wakefulness and sleep; however, the frontalis exhibits substantially less between-subject variability. This study established face validity for the use of *f*EMG in the detection of wake and stages of sleep, and for future applications toward assessment of quantitative REM sleep muscle activity in REM sleep behavior disorder.

Keywords: EMG, frontalis, submentalis, sleep stage, REM sleep without atonia

Introduction

Acquisition of submental electromyographic activity (*s*EMG) is recommended in the guidelines of the American Academy of Sleep Medicine for laboratory polysomnography (PSG).[1] The *s*EMG signal is used to assist with visual identification of sleep onset, micro-arousals, rapid eye movement (REM) sleep, and REM sleep without atonia (RSWA).[2] Changes in sleep stage can be recognized by modulations in *s*EMG amplitude occurring across relatively wide time windows. *s*EMG has also been shown to be highly sensitive and specific for the recognition and quantification of phasic and tonic muscle activity in REM sleep behavior disorder (RBD). *s*EMG additionally aids in the distinction between pathologic RSWA diagnostic of RBD and physiologically increased REM sleep muscle activity due to respira-

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tory arousals in obstructive sleep apnea (OSA) patients, as well as from normative RSWA levels in patients with OSA without RBD.[3–8]

Frontalis EMG power (*f*EMG), derived from frontopolar electroencephalographic (EEG) activity, has been used for the automated staging of sleep and to visually confirm autostaged records.[9–12] The display of *f*EMG power relative to the alpha, sigma, and beta power, when used during a focused technical review, contributed to a 5% improvement in the staging of awake (ie, from 82% to 87%) and a 7% improvement in the staging of REM sleep (ie, from 73% to 80%).[9] It has also been demonstrated that EMG activity in the frontalis muscles can identify subclinical abnormalities associated with neurodegenerative disease.[13]

This study was designed to assess the concordance between *s*EMG and *f*EMG power with the goal of establishing feasibility and face validity for the use of *f*EMG power as an alternative to *s*EMG in a self-applied device that can stage sleep without the chin derivation.

Methods

Recordings

Forty-three records with simultaneous acquisition of differential signals from the submental and frontalis muscles with at least 3 hours of PSG recording time were selected for this study. The records were acquired with patient consent under the approval of the Alpha IRB (IRB00006205, protocol 201501) at Complete Sleep Solutions in Murrieta, California, a facility accredited by the American Academy of Sleep Medicine (AASM). Thirty-six records were from subjects referred for a PSG for an assessment of sleep-disordered breathing, and seven were from healthy controls.

The *s*EMG signals were recorded during laboratory PSG according to AASM criteria using Alice 5 systems (Philips Respironics, Monroeville, PA, USA). The *f*EMG was extracted from a simultaneously recorded EEG signal acquired with the Sleep Profiler (Advanced Brain Monitoring, Inc., Carlsbad, CA, USA) from sensors located at AF7 and AF8, sampled at 256 s/s. Figure 1 depicts the location of the Sleep Profiler when worn; written informed consent was obtained from the subject for use of her image. A detailed description of the Sleep Profiler data device and its application during data acquisition was previously provided.[9]

Signal processing

The *s*EMG signal was transferred from the PSG record, upsampled using sample repetition to 256 s/s and saved into the Sleep Profiler record. The appearance of the primary signals used for this study is presented in Figure 2. The *s*EMG and



Figure 1 Subject wearing the sleep profiler.

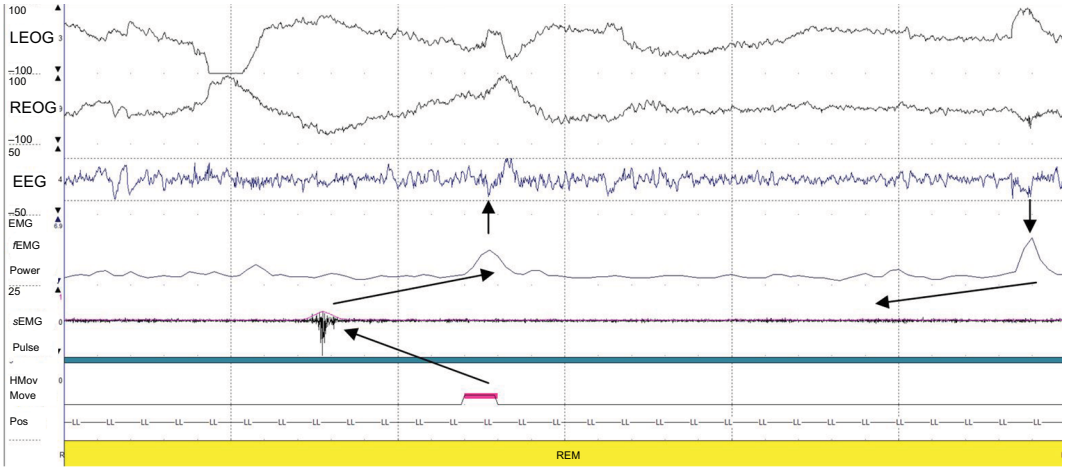

Figure 2 Thirty seconds epoch during REM sleep with differential recordings from AF7-Fpz, AF8-Fpz, and AF7-AF8 (labeled LEOG, REOG, and EEG, respectively), *f*EMG power, differential EMG, head movement, and head position.

Notes: The first arrow highlights the 4.5 seconds misalignment that coincided with a movement ~2.5 hour into the study. The second arrow identifies a muscle twitch (eg, facial muscle contraction), based in its appearance in the frontopolar signals but not the submental EMG signals.

Abbreviations: EMG, electromyography; *f*EMG, frontalis electromyography; REM, rapid eye movement.

*f*EMG signals were identically filtered with a 40 Hz high-pass IIR filter with 61 Hz, –3 db roll off to derive EMG power (μ^2 at 1 second epochs). The *s*EMG and *f*EMG power values were averaged using a 5 second window (2 seconds before and 2 seconds after the current sample). Periods were excluded when the PSG power equaled zero (ie, when disconnected during the restroom breaks). One value of *f*EMG power was then removed every 30 minutes to adjust for misalignment attributed to differences between the hardware clocks. The effectiveness of this transformation was confirmed by comparing values from the first half of each 30 minutes period to the second half of each period.

All studies were visually inspected for artifact that could impact the results; a total of four records were excluded. Two records were excluded due to technically inadequate EEG signal that affected the *f*EMG power. One record was excluded due to synchronization issues caused by excessive signal loss in the *s*EMG signal, and another due to uncharacteristic *f*EMG contamination attributed to breathing artifact (Supplementary material). The descriptive statistics in Table 1, when applicable, were derived from the PSG data.

Data reduction and analyses

CC analysis was used to evaluate the temporal relationship between absolute changes in *s*EMG in comparison with *f*EMG power. CC across 1 second epochs was used to measure the concordance between *s*EMG and *f*EMG power. Next the *s*EMG and *f*EMG power values were averaged to 15 minute intervals prior to CC analysis to evaluate individual differences in agreement after smoothing for variability related to sleep stage, microarousals and sleep disordered breathing.

Sleep staging was obtained from the previously reported majority agreement across five raters.[9] Each record's 1 second *f*EMG and *s*EMG power values associated with a consensus sleep stage were averaged. CC was applied by-subject to the mean power across wake, N1, N2, N3, and REM. In two cases REM sleep was not observed, and in two records N3 was absent. Apnea-hypopnea indexes (AHIs) were scored by one rater, based on AASM 2012 criteria with a 3% desaturation.[1]

Pearson correlation was used to evaluate the relationship between CC values, sleep efficiency, and AHI. Correlations were stratified by strong (>0.70), moderate (>0.70 and ≥0.50), and weak (<0.50) agreement. *t*-tests were used to identify significant differences in by-stage EMG power.

Results

Concordance across 1 second and 15 minute intervals

When the 1 second absolute power values were compared, the mean CC across all records was 0.63; 36% of the records exhibiting a strong agreement, 49% showing moderate agreement, and 15% displaying weak agreement (Table 2). With the EMG power values averaged into 15 minute increments, the mean CC across all increments in the study was 0.79. The agreement between *s*EMG and *f*EMG power was strong in 77% of the records, moderate in 18% of the records, and weak in 5% of the records (Table 2). There was a moderate association between the AHI and mean 15 minutes CC values (r=0.52, P=0.001), but not with 1 second CC values.

Concordance by and across sleep stages

Distributions of the mean across-subject power are presented by stage in Table 3 with the coefficients of variation. Mean *f*EMG power was greater and with at least 50% less variability compared to *s*EMG power during wake and across sleep stages. EMG power progressively decreased for both signals as NREM sleep deepened.

N1 mean power was significantly greater than the REM power for both *s*EMG and *f*EMG (P=0.004 and 0.0001, respectively). *s*EMG and *f*EMG power during N1 was at least 30% greater than that during REM in 87% of the cases (32/37), and both are less in 8% of the cases (3/37). While *s*EMG power during REM was significantly less than *f*EMG power, the variability about the mean was four times greater.

Table 1 Descriptive statistics

Subjects, n	39
Males, n (%)	29 (74)
Age, years, mean (SD)	44±14
Recording time, min, mean ± SD	371±84
Sleep efficiency, %	77±13.7
Non-REM time; %	83±7
REM time, %	17±7
AHI, events/h	16±20

Abbreviations: AHI, apnea-hypopnea index; REM, rapid eye movement.

Table 2 Distributions of mean CC results stratified by strength of agreement when 1 second and 15 minutes averaged *s*EMG and *f*EMG power was compared

EMG power increment	Strong		Moderate		Weak	
	n	CC	N	CC	n	Mean CC
1 second	14	77±4.4	19	62±6.2	6	34±10.7
15 minutes	30	87±7.6	7	64±6.1	2	10±40.3

Abbreviations: CC, cross-correlation; EMG, electromyography; *f*EMG, frontalis electromyography; *s*EMG, submentalis electromyography.

Table 3 Distributions of power (μV^2) and CV across all records by stage

	*s*EMG		*f*EMG		
Stage	**Mean ± SD**	**CV (%)**	**Mean ± SD**	**CV (%)**	***P*-value**
Wake	875±974	111	1501±794	53	<0.01
N1	449±450	100	559±243	44	0.18
N2	292±267	92	325±117	36	0.48
N3	256±194	76	268±101	38	0.73
REM	216±138	64	279±42	15	0.01

Abbreviations: CV, coefficient of variation; EMG, electromyography; *f*EMG, frontalis electromyography; REM, rapid eye movement; *s*EMG, submentalis electromyography.

As expected, *s*EMG power was lower during REM sleep; however, the lowest mean *f*EMG power was observed during N3. There was a moderate concordance between the 37 subjects' *f*EMG and *s*EMG power values during stage N3 (r=0.49, P=0.002), likely the result of there being only a 4% difference in the mean power. The by-stage magnitude of *f*EMG and *s*EMG power was unaffected when stratified by age, sex, or OSA (ie, AHI >10). Both *s*EMG and *f*EMG power values when awake were significantly greater than the respective mean power values during N1 (P=0.02 and <0.0001, respectively). *s*EMG power during N1 was significantly greater than the mean power of N3 and REM (P=0.02 and <0.01, respectively). *f*EMG during N1 was significantly greater than the mean power of N2, N3, and REM (all P<0.0001), and *f*EMG power during N2 was significantly greater than REM (P=0.03).

Relative changes in *s*EMG and *f*EMG power across wake, NREM, and REM stages for each subject were essentially identical. The median CC was 0.98 with interquartile CC ranges of 0.97 and 0.99. The gross outlier (CC=0.27) had an AHI of 109 events/hour and was one of four cases when the *s*EMG power was greater than *f*EMG power across all stages of NREM sleep.

Discussion

We found that temporal changes in EMG measured from the frontalis muscle are highly similar to the submentalis muscle. There was strong concordance between the *s*EMG and *f*EMG power when smoothed at 15 minute intervals, ie, 95% of the records exhibited at least moderate agreement, suggesting a trait-like relationship between the two signals during sleep and wake. The 1 second CC confirmed that the signals were measuring similar information but the relationship was influenced by micro-arousals and variability in EMG power during awake periods.

During wake periods, *f*EMG power exhibited 50% greater power and 50% less across-subject variability, suggesting its superiority to *s*EMG for purposes of differentiating sleep and wake. This finding likely resulted from differences in inter-electrode spacing (ie, 11.3 vs ~4 cm). The across-subject variability in *s*EMG power, being two to four times greater than *f*EMG power across all stages of NREM and REM, may be attributed to differences in the number of motor units in the frontalis vs submentalis muscles. This explanation cannot be easily confirmed because it is difficult to accurately assess motor unit number in the facial muscles, because the muscles are difficult to activate in isolation.[14] Alternatively, the divergence in adipocyte distributions over the submentalis muscles, which attenuate the EMG signal, may contribute to the increased across-subject variability.[15]

In clinical practice, *s*EMG is most often useful in the differentiation of N1 or drowsy wakefulness from tonic REM and to identify bursts of phasic as well as tonic muscle activity in patients with RBD.[16] We found that the magnitude of both *s*EMG and *f*EMG was at least 30% greater in N1 as compared to REM in 87% of the subjects. The influence of sleep stage on EMG power was more apparent in the frontalis recordings. While mean *s*EMG power during N1 was significantly greater than REM and N3, *f*EMG power during N1 was significantly greater than REM, N2, and N3, while REM was significantly lower than N2. The difference in EMG power transitioning from wake to sleep onset was also more clearly defined in the *f*EMG signal. These findings suggest that *f*EMG is at least non-inferior to *s*EMG for use in the staging of sleep in patients without REM-related sleep disorders.[9–12] Whether an EMG signal with greater stage-related changes in power impacts the detection of RSWA is yet to be demonstrated.

No significant differences in power were observed for either *f*EMG or *s*EMG when stratified by sleep stage, age, sex, and sleep disordered breathing, unlike what was reported for waking state EEG.[17] As expected, muscle tone in the submentalis decreased on a continuum and was lowest during REM. The frontalis exhibited a similar decline; however, the cross-subject mean power during REM was slightly greater as compared to N3. One possible explanation for this relative increase in frontalis hyperactivity during REM may be

a result of facial muscle contractions. Rivera-García et al suggested this increased REM-specific phasic and sustained facial muscle activity was facilitated by limbic activity and may be related to higher emotional activity, a condition that might be exacerbated by RBD.[18] Despite this potential trait-like influence, it is important to note that the frontalis was less prone to individual differences during REM and exhibited four times less between-subject variability as compared to the submentalis.

Detection of RSWA has been extensively investigated using different muscles and different methods (ie, biceps brachii, extensor digitorum brevis, flexor digitorum superficialis, mentalis, and sternocleidomastoid muscles).[2–7,19,20] RSWA in the submentalis has been shown to predict the subsequent development of a defined neurodegenerative disease in patients with idiopathic RBD and to correlate with disease severity in Parkinson's disease.[21] Ferri et al have also reported that the submentalis muscle provides an excellent EMG measure for automated detection of RBD, with subsequent corroboration of automatic and "gold standard" visual RSWA methods.[3,4,22] It will be interesting to evaluate patients with RBD compared to controls to determine if frontalis muscle activity was overlooked as a potential contributor to future automated methods for the detection of RSWA.

Next to be demonstrated is whether the strong CC between *f*EMG and *s*EMG, presumably driven by muscle activity resulting from spontaneous, movement-related, and/or respiratory arousal artifact will be apparent in: 1) what is quantified as RSWA for the diagnosis of RBD ie, phasic muscle activity during REM sleep, 2) normal community and sleep apnea controls without dream enactment, or 3) patients receiving antidepressants.[2,23–25] Studies are currently underway in the USA and Europe to investigate the relationship between *f*EMG and *s*EMG, as well as EMG signal recorded from the arms and legs in patients with idiopathic REM behavioral disorder, to determine if *f*EMG has sufficient diagnostic accuracy for use in large epidemiological studies for home-based RSWA screening.

Conclusion

The strong concordance between the *s*EMG and *f*EMG temporal power confirms feasibility and face validity for the use of *f*EMG in the detection of wake and stages of sleep. The transition from wake to sleep as well as sleep stage-related changes in power were more apparent in the frontalis EMG signal. *s*EMG power was significantly less than *f*EMG during REM, however, its across-subject variability was four times greater. No significant differences in power were observed for either *f*EMG or *s*EMG during sleep when stratified by stage and by age, sex, and sleep disordered breathing.

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Disclosure

Coauthors Levendowski, Westbrook, and Berka would benefit financially if the Sleep Profiler intellectual property was acquired by a third party. The authors report no other conflicts of interest in this work.

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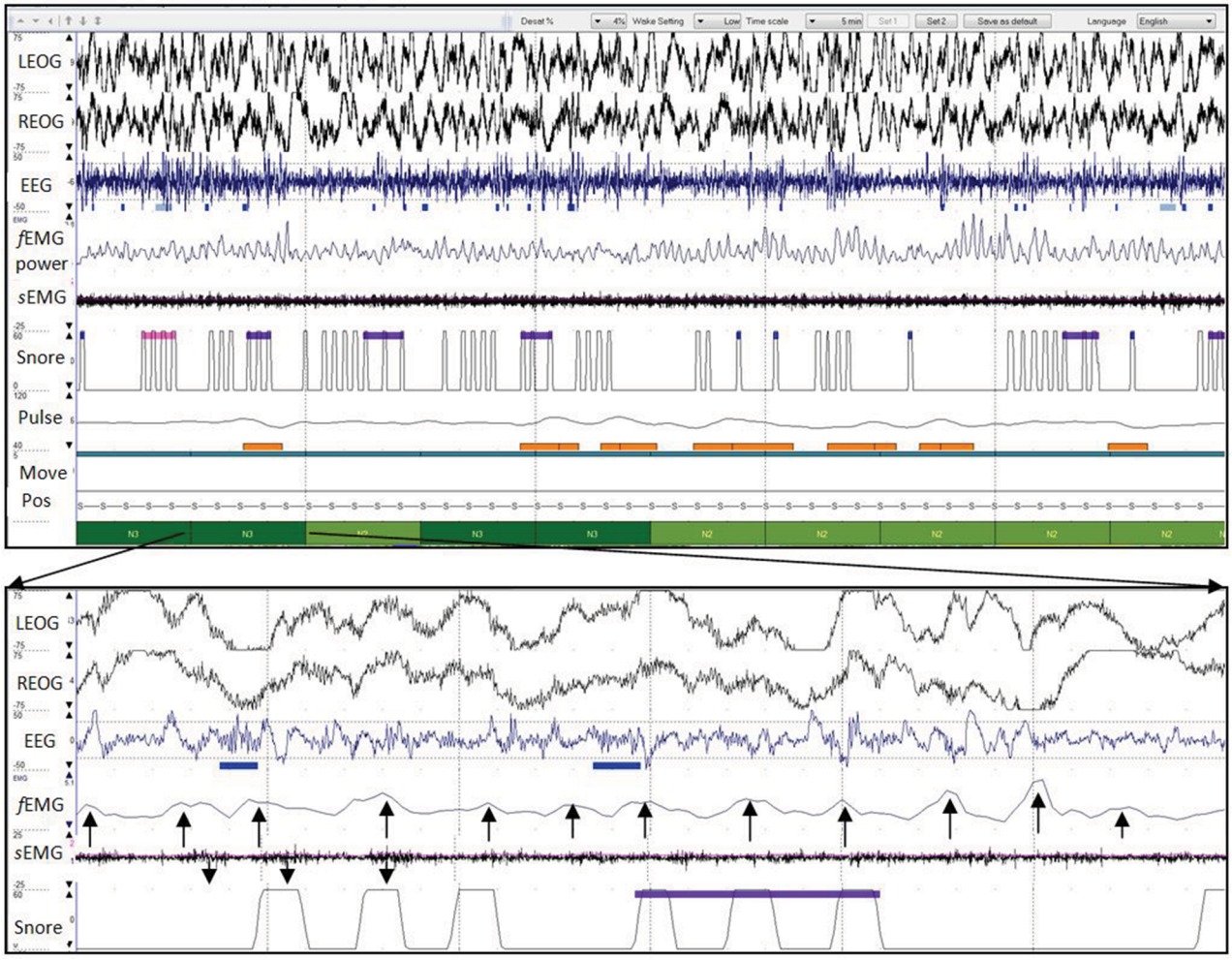
Supplementary material

The screenshots below present the signal characteristics for a 5 minute epoch and an excerpted 30 second epoch. In both the screenshots, there is a cyclical drift in the LEOG and REOG channels associated with sweat artifact, a pattern that occurs during non–rapid eye movement sleep and when the headband is not properly affixed.

The frontalis electromyography (*f*EMG) power (extracted from the electroencephalographic signal) shows pulsatile changes timed to breathing (likely associated with changes in venous pressure) while the submental EMG signal shows minimal changes in amplitude.

In the lower 30 second screenshot, the changes in *f*EMG power are timed to snoring and suggest a breathing rate of 24 bpm. There are subtle increases in submentalis electromyography (*s*EMG) amplitude during some of the periods with loud snoring but the concordance between *f*EMG and *s*EMG is compromised.

The automated sleep staging algorithms compensates for large amplitude breathing-related EMG when it occurs in conjunction with loud snoring; however, auto-staging requires a more detailed technical review when the puslating *f*EMG patterns are apparent.



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