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Polysomnographic Plethysmography Excursions are Reduced in Obese Elderly Men*

Magnus Ruud Kjær[1,2,3], Andreas Brink-Kjær[1,2,3], *Member, IEEE*, Umaer Hanif[1,2,3], *Member, IEEE*, Emmanuel Mignot[3,×], Poul Jennum[2,×], and Helge B. D. Sørensen[1,×], *Senior Member, IEEE*

Abstract— **Sleep apnea is a widespread disorder and is defined by the complete or partial cessation of breathing. Obstructive sleep apnea (OSA) is caused by an obstruction in the upper airway while central sleep apnea (CSA) is characterized by a diminished or absent respiratory effort. It is crucial to differentiate between these respiratory subtypes as they require radically different treatments. Currently, diagnostic polysomnography (PSG) is used to determine respiratory thoracic and abdominal movement patterns using plethysmography belt signals, to distinguish between OSA and CSA. There is significant manual technician interrater variability between these classifications, especially in the evaluation of CSA. We hypothesize that an increased body mass index (BMI) will cause decreased belt signal excursions that increase false scorings of CSA. The hypothesis was investigated by calculating the envelope as a continuous signal of belt signals in 2833 subjects from the MrOS Sleep Study and extracting a mean value of each of the envelopes for each subject. Using linear regression, we found that an increased BMI was associated with lower excursions during REM sleep (-0.013 [mV] thoracic and -0.018 [mV] abdominal, per BMI) and non-REM (-0.014 [mV] thoracic and -0.012 [mV] abdominal, per BMI). We conclude that increased BMI leads to lower excursions in the belt signals during event-free sleep, and that OSA and CSA events are harder to distinguish in subjects with high BMI. This has a major implication for the correct identification of CSA/OSA and its treatment.**

I. INTRODUCTION

Sleep Apnea (SA) is defined as the presence of events with complete (apnea) or partial (hypopnea) cessation of breathing during sleep and is divided into different subtypes: Obstructive sleep apnea (OSA), which occurs when an obstruction in the upper airway prevents the flow of air; central sleep apnea (CSA), which has a neurological component that causes a diminished or absent effort to breathe (e.g., due to cardiac disorders, brain disease or treatment with respiratory depressant medication); and Mixed Sleep Apnea, which contains both OSA and CSA events. Both OSA (most prevalent) and CSA (less prevalent) result in complete cessation of breathing during sleep [1]. As the pathology and treatment of OSA and CSA are different, correct classification is crucial. Currently, a diagnostic polysomnography (PSG) is used to diagnose SA by manually scoring the events based on the respiratory pattern as determined by nasal pressure and oral thermal sensors, thoracic and abdominal excursions, blood oxygen saturation (SpO2), and snoring measured by a piezoelectric sensor. There are several methods for measuring thoracoabdominal respiratory movements including piezoceramic elements, plethysmography, and respiratory inductive plethysmography (RIP) [2]. Although newer methods such as radar signals have shown potential [3], RIP remains the gold standard in clinical practice. In RIP, the belt is made of elastic bands and contains insulated coils, which are sewn into the elastic bands, and this is the method used for monitoring respiratory movement in the MrOS study from which data is used in this paper. The inductance through the coils is measured and as bands stretch during respiratory movement the coils change and due to the constant current, the magnetic flux changes proportionally to the velocity of change in the band length. The signal is demodulated and an amplified voltage level relative to the respiratory band movement is created [4]. Respiratory movements determined from PSG are central for differentiating between OSA and CSA. The underlying causes of OSA and CSA present themselves differently in plethysmography belt signal excursions, as during a CSA event there is a lack of respiratory effort resulting in an absence of thoracoabdominal excursions in the belt signals, while during an OSA event there is increased respiratory effort to compensate for the cessation of breathing which is reflected by excursions in the belt signals [1]. The different excursions for OSA and CSA events have been visualized in Fig. 1 There is significant interscorer variability in the evaluation of SDB, especially in differentiating OSA from CSA [5]. As OSA is treated with continued positive airway pressure (CPAP), surgery, or weight reduction and CSA is managed by use of adaptive serve ventilation (ASV) it is important to correctly differentiate between the patterns. The greatest contributing factor for developing SDB is overweight. In this paper we hypothesize that an increased body mass index (BMI) will lead to decreased excursions in the plethysmography belt

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[1]Department of Health Technology, Technical University of Denmark, 2800 Kgs. Lyngby, Denmark, s154555@student.dtu.dk

[2]Danish Center for Sleep Medicine, Rigshospitalet, 2600 Glostrup, Denmark

[3]Stanford Center for Sleep Sciences and Medicine, Stanford University, Palo Alto, CA 94304, USA

[×]Shared last authors

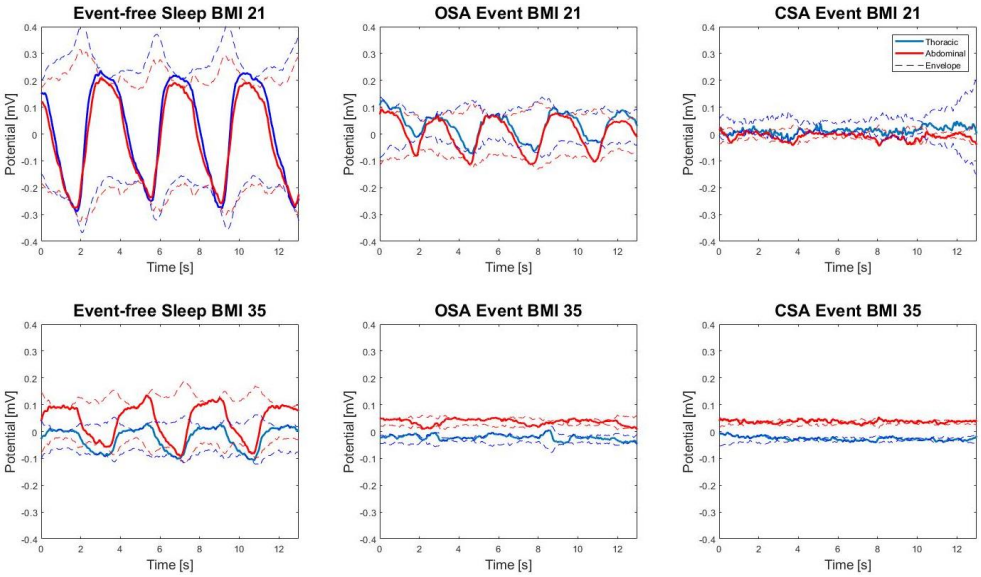

Figure 1. Visualization of *RIP* belt signals (*solid*) and coherent envelopes (dotted) for a subject with BMI = 21 (top row) and a subject with BMI = 35 (bottom row) during event-free sleep (left column), obstructive sleep apnea (OSA) event (middle column), and central sleep apnea (CSA) event (right column) in thoracic (blue) and abdominal (red) channels.

signals recorded during sleep. This is of clinical interest due to RIP being widely used to distinguish between obstructive and central events during sleep. The study examines the RIP belt signals (henceforth known as belt signals) of 2883 subjects from the MrOS cohort.

II. Data Description

The MrOS (Osteoporotic Fractures in Men Study) was recorded between December 2003 and March 2005 on 2911 subjects. Data from the study includes complete PSG measurements, however, this paper will focus merely on the belt signals along with measurements of height and weight that were used to derive BMI. Subjects in the study range from 67 years old to more than 90 years old (76.35 ± 5.47) and were all male. The Summit IP RIP was used to record belt signals during sleep for all subjects [4], [6], [7].

III. Methods

The data processing was done using MATLAB R2020b. The envelope of each complete belt signal was found using the Hilbert transformation with the approximate analytical signal using the algorithm first introduced in [8]. Hereafter the difference of the upper and lower signal envelope was calculated as a continuous measure describing the amplitude of belt signal excursions as seen in Fig. 1. Next, using manual annotations, we subdivided each subject's amplitude of belt signal excursions, into the event types: CSA, OSA, HYPO, NONE, and ALL. The NONE event was defined as 10 seconds away from any SA event to exclude recovery breaths. We further divided these regions based on sleep stages: rapid eye movement (REM), non-REM (NREM), and all sleep. For example, the mean excursion under CSA events during REM is denoted $\mu_{CSA\text{-}REM}$. These measures allow us to investigate the hypothesis: an increased BMI will lead to decreased belt signal excursions. The investigation was carried out by calculating a linear regression coefficient between mean excursions, and BMI. Experiments were conducted on measures: REM, NREM, NONE, and derived measures such as the percentage change of CSA events compared to NONE during REM per BMI described by:

$$P_{\frac{CSA}{NONE}REM} = 100 \cdot \left(1 - \frac{\mu_{CSA\text{-}REM}}{\mu_{NONE\text{-}REM}}\right) \quad (1)$$

Equation 1 allows the investigation of comparative changes from baseline (NONE) and CSA, which is clinically important as the changes from baseline determines the annotation of each event. Furthermore, the linear regression was done on the percentage change as calculated in equation 1 and reported in Table 1. This allows for a more intuitive understanding of the effects of increasing BMI on belt signal excursion. Moreover, the hypothesis that the mean excursions of belt signals is lower for obese subjects (≥ 30 BMI). This hypothesis is shown below:

$$H_0: \mu_{BMI<30} = \mu_{BMI>=30}$$
$$H_a: \mu_{BMI<30} > \mu_{BMI>=30} \quad (2)$$

Where $\mu_{BMI<30}$ is the mean excursions of belt signals during sleep of subjects with a BMI below 30 and $\mu_{BMI>=30}$ is the same of subjects with a BMI above or equal to 30. To test the null hypothesis, a two sample one sided t-test was conducted. The goodness of fit was calculated on all measures versus BMI to ensure that samples were part of a normal distribution. Many experiments were conducted which necessitate correction for multiple testing, which was done using the Benjamini & Hochberg procedure [9].

IV. Results

In Fig. 1 an example of a belt signal is shown for a subject with a BMI of 21 (S21) and a subject with a BMI of 35 (S35). Here, both signals are shown outside breathing events while sleeping. The continuous amplitude of the excursions is shown as the envelope of the signal found via the Hilbert transformed described in the method section above. The belt signal excursions of S35 in this example has much lower amplitude both in the thoracic and abdominal channel than the corresponding signal from S21. Furthermore, the thoracic belt signal of S35 is very similar to the signal found in

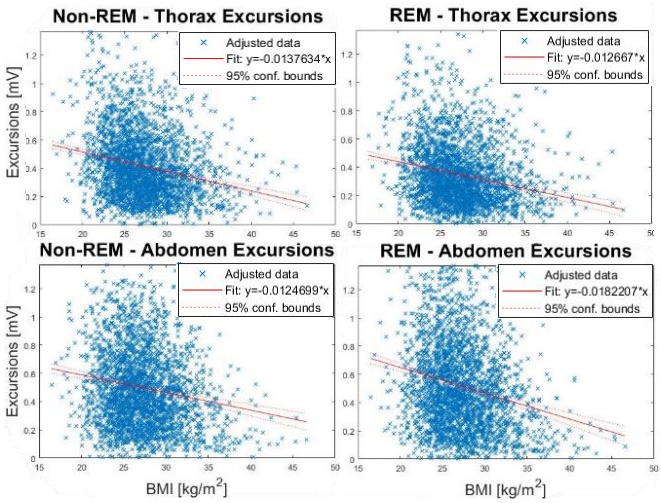

Figure 2. Scatterplots of average belt signal excursions during event-free non-REM and REM sleep against their pertaining BMI in the abdominal and thoracic channels. Furthermore, on each scatter plot a linear regression model has been fitted that show the overall trend of belt signal excursions per BMI. A total of 2883 datapoints were used from 2883 PSGs to create the linear regression, however, some were left out of the plot to make it more understandable. (Top Left: 18 datapoints not shown; Top Right: 17 datapoints not shown; Bottom Left: 49 datapoints not shown; Bottom Left: 60 datapoints not shown.)

Fig. 1 during a CSA event. In Fig. 2 we present the average belt signal excursions during event-free sleep of all 2883 subjects against their pertaining BMI. Across wake, non-REM, and REM, a significant trend in the data is found via linear regression. The average percentage change of belt signal excursions per BMI for OSA, CSA, and HYPO are detailed in Table 1. Here, a significant negative change in belt signal excursions per BMI was seen during non-REM sleep while having OSA events in both the abdominal and thoracic channels, a negative change was only observed for the thoracic channel while having CSA events and the abdominal channel while having HYPO events. In Table 2, the hypothesis seen in (2) is tested and results are reported. The significant experiments seem to occur during event-free sleep and HYPO during both REM and NREM, and OSA during REM sleep.

Table 1. Average percentage change of belt signal excursions per BMI is shown for obstructive sleep apnea (OSA), central sleep apnea (CSA), and hypopnea (HYPO). Bold indicates that the null hypothesis was rejected using the Benjamini & Hochberg procedure [9]. A: abdominal belt; T: thoracic belt, PC: percent change.

Event	Stage	Modality	PC / BMI	P-value
OSA/ NONE	Non-REM	A	**-1.2908**	**2.34e-12**
		T	**-1.3418**	**8.72e-10**
	REM	A	**-0.4530**	**0.025**
		T	**-0.9224**	**0.91e-02**
CSA/ NONE	Non-REM	A	**-1.0480**	**6.00e-07**
		T	-0.5655	0.085
	REM	A	**-0.5617**	**0.014**
		T	-0.4653	0.063
HYPO/ NONE	Non-REM	A	-0.1633	0.12
		T	**-0.5473**	**1.62e-07**
	REM	A	0.0899	0.40
		T	**-0.2750**	**0.014**

V. Discussion

As shown in Fig. 2, there is a large significant drop in belt signal excursions associated with increased BMI. Hereby we conclude that our initial hypothesis that an increase in BMI will cause decreased belt signal excursions is correct for this dataset. Furthermore, Fig. 2 reveals a high variance between average excursions during sleep for subjects across all BMI values. This is a sign of poor data-quality that may lead to confusion regarding differentiating CSA and OSA events. Moreover, the estimated regression coefficients for thoracic (REM: 0.013[mV], NREM: 0.014[mV]) and abdominal (REM: 0.018[mV], NREM: 0.012[mV]) excursions show similar estimations across REM and NREM sleep along with the thoracic and abdominal modalities. In Table 1 a large drop of belt signal excursions during OSA events as a fraction of coherent event-free sleep is reported across BMI with a drop of 1.29% and 1.34% per BMI during non-REM sleep and 0.45% and 0.92% during REM sleep in the abdominal and thoracic channels, respectively. A similar trend is shown for CSA events as a fraction of the coherent event-free sleep, although this is only significant for the abdominal channel showing 1.0480% decrease of belt excursions per BMI during non-REM and 0.5617% decrease during REM. Both findings indicate that it will be more difficult to see changes in belt signals from event-free sleep to OSA and CSA events in subjects with high BMI as clearly visualized in Fig. 1. Complex breathing, which is the persistence of CSA after CPAP treatment of OSA, is often seen in patients [10]. It would be clinically relevant to investigate patients prior to CPAP treatment and investigate the presence of CSA and OSA components in the apneic events as well as evaluate the respective components more clearly after CPAP treatment. In Table 2 a significant drop in belt signal excursions is seen during event-free sleep, HYPO events (REM and non-REM), and OSA events (REM) from subjects under 30 BMI (BMI30-) to subjects with a BMI above or equal to 30 (BMI30+). The lower excursions for BMI30+ is carried throughout all events, apart from CSA which is expected, and makes it more likely that a low excursion during OSA will lead to misclassification of OSA as CSA. The significant drop during REM under OSA events can be interpreted as BMI30+ compensating with muscles during non-REM sleep but not being able to access these voluntarily contracting muscles during REM sleep as these are paralyzed, thus not being able to compensate. This observation is likely related to Obesity Hypoventilation Syndrome (OHS). OHS is when obesity leads to hypoventilation partially due to extra weight placed on the chest in the form of fat and partially due to excess fat producing hormone and altered brain function that decreases ventilation efficiency [11]. This explanatory study poses questions regarding data quality and the patterns described above leads to some uncertainties in distinguishing between event-free, OSA, and CSA events with increased BMI. This would in turn hinder artificial intelligence (AI) in learning the underlying truth in the signals based on technician-scorings, causing the AI to incorrectly classify CSA and OSA events and create much confusion between these. Finally, it is considered that the MrOS datset

Table 2. Differences in belt signal excursions in subjects with a BMI less than 30 (BMI30-) and over or equal to 30 (BMI30+). Bold indicates rejection of the null hypothesis using the Benjamini & Hochberg procedure [9] and thus that the BMI30- group is significantly different to BMI30+ with regards to average belt signal excursions. EF: Event-Free, A: abdominal belt, T: thoracic belt, E:event, M: modality

E	Stage	M	BMI 30- ($\mu \pm \sigma$)	BMI 30+ ($\mu \pm \sigma$)	P-value
EF	N-REM	A	**0.52± 0.32**	**0.42± 0.31**	**1.34e-11**
		T	**0.43± 0.25**	**0.36± 0.24**	**2.67e-11**
	REM	A	**0.55± 0.35**	**0.41± 0.32**	**1.06e-18**
		T	**0.36± 0.27**	**0.29± 0.21**	**1.65e-11**
OSA	N-REM	A	0.28± 0.22	0.27± 0.22	0.19
		T	0.23± 0.19	0.22± 0.17	0.060
	REM	A	**0.28± 0.21**	**0.23± 0.25**	**0.12e-02**
		T	**0.20± 0.16**	**0.17± 0.16**	**0.013**
CSA	N-REM	A	0.21± 0.17	0.20± 0.17	0.22
		T	0.16± 0.16	0.15± 0.13	0.060
	REM	A	0.20± 0.17	0.18± 0.21	0.14
		T	0.13± 0.10	0.13± 0.12	0.28
HYPO	N-REM	A	**0.36± 0.24**	**0.30± 0.23**	**6.54e-09**
		T	**0.31± 0.20**	**0.27± 0.20**	**1.01e-05**
	REM	A	**0.38± 0.25**	**0.27± 0.25**	**3.87e-19**
		T	**0.23± 0.18**	**0.19± 0.15**	**8.21e-10**

is all male and therefore the further investigation is necessary for broader populations. We argue that populations of men and women should be investigated separately as both hormonal (before menopause) and anatomic differences can cause impactful differences in results for male and female populations.

VI. Conclusion

We can conclude that the respiratory movement plethysmography belt signals recorded in a polysomnography are lower in amplitude with subject of higher BMI during event-free REM sleep (-0.013 [mV] thoracic and -0.018 [mV] abdominal, per BMI) and NREM (-0.014 [mV] thoracic and -0.012 [mV] abdominal, per BMI). This indicates that less information is carried in the amplitude part of the excursion signal as BMI increases. Moreover, it has been shown that for 2833 patients in the MrOS dataset being obese yields significantly lower excursions in the belt signals during event-free sleep (REM and NREM) and for OSA (REM) but not for CSA. Therefore, in obese patients it is more likely that an OSA event will be classified as an CSA event.

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