THE CORRELATION ANALYSIS BETWEEN AIRFLOW AND OXYGEN SATURATION IN SLEEP APNEA EVENTS

Necmettin Sezgin[a], Mehmet Akin[a], Gokhan Kirbas[b]
necmettinsezgin@gmail.com, makin@dicle.edu.tr, gkirbas@dicle.edu.tr
a. Department of Electrical and Electronics Engineering, Dicle University, Diyarbakir, Turkey.
b. Faculty of Medicine, Dicle University, Diyarbakir, Turkey

ABSTRACT

Diagnosis of Sleep apnea is currently performed by a full night polysomnography study at sleep laboratories. The majority of apnea patients are treated by constant positive airway pressure (CPAP) device. In this paper, we aim to find a correlation between airflow and oxygen saturation (SaO_2) in sleep apnea events. The apnea can be broadly classified into three types: obstructive sleep apnea (OSA), central sleep apnea (CSA) and mixed sleep apnea (MSA). During OSA, the airway is blocked while respiratory efforts continue. During CSA the airway is open, however there are no respiratory efforts. There is an efficient correlation between airflow and SaO_2 in sleep apnea events. In this paper, it is aimed to find the correlation degree between airflow and oxygen saturation in sleep apnea events. In the future studies the correlation will help to detect the sleep apnea such as an intelligent system.

Keywords: Correlation, SAS, Airflow, SaO2

I. INTRODUCTION

Sleep apnea syndrome (SAS) is characterized by repeated temporary cessation of breathing to the lungs during sleep, which is known as periods of apnea [1]. Clinically, apnea is defined as complete cessation of breathing for more than 10 seconds in adults [2]. Normally, when a person is awake, the upper airway remains open, permitting airflow to the lungs, except for momentary closures during swallowing and speech [3]. In some periods, however, the throat lumen may become obstructed during sleep [4]. It can be broadly classified into three types: Obstructive sleep apnea (OSA), Central sleep apnea (CSA) and Mixed sleep apnea (MSA). During OSA the airway is blocked while respiratory efforts continue. During CSA the airway is open, but there are no respiratory efforts. However, the mechanisms underlying these different types of sleep apnea are likely to overlap [5]. The prevalence of sleep-disordered breathing is approximately 2% in women and 4% in men who are between 30 and 60 years of age [6]. The majority of patients with SAS are diagnosed with OSA, the most common form of sleep apnea. Physical obstruction of the airway can result from a variety or combination of anatomical factors [7] such as enlarged tonsils [8], enlarged uvula [9], increased tongue size [10] and abnormal craniofacial morphology [11]. Genetics have also been found to be a factor [12-14]. In individuals with OSA, numerous sleep-related obstructive breathing events can occur throughout the night. In mild cases, there can be 5–15 episodes per hour and in severe cases more than 30 episodes per hour [15]. These respiratory disturbances may lead to hypoxia and hypercapnia, which can trigger arousal from sleep by increasing ventilatory drive [16-18]. As a result of such sleep disruption, excessive daytime sleepiness is the most common presenting complaint [19]. Other symptoms of sleep apnea may include loud snoring, not feeling well-rested in the morning, chronic fatigue [20], falling asleep at inappropriate times of day, morning headaches, recent weight gain, limited attention span, memory loss, poor judgment, personality changes, and lethargy [21]. These symptoms can significantly decrease quality of life and increase the risk of accidents [22, 23].

Unfortunately, sleep apnea may go undiagnosed for years [24, 25]. This is most likely because the people themselves may not remember the events of apnea. For this reason, it is often the patient's spouse, roommate or family member who may witness the periods of apnea, alternating with arousals and accompanied by loud snoring [26, 27]. However, it is important to note that although snoring is the most common complaint associated with sleep apnea, most patients who snore do not have sleep apnea [26, 28]. Therefore, patients reporting symptoms of SAS should be referred to a sleep center for an overnight sleep study. SAS is usually diagnosed by overnight polysomnography, including simultaneous electroencephalography (EEG), electromyography (EMG), electrocardiography (ECG), electro-oculogram (EOG), oximetry, airflow through the mouth and nose, thoracic and abdominal respiration by plethysmography [29]. From the overnight sleep study, the number of obstructive breathing events per hour can be determined. This calculation is commonly called the respiratory disturbance index (RDI) or more specifically, the apnea-hyponea index (AHI), which represents the sum total of apneas, hypopneas and respiratory arousals per hour of sleep [30]. The RDI value is used to diagnose and grade the severity of the sleep apnea [15]. AHI has been used frequently to assess the severity of apnea, according to the Chicago criteria: AHI < 5 (normal), AHI = 5–15 (mild), AHI = 15–30 (moderate), and AHI > 30 (severe) [31]. Risk factors for SAS include upper airway abnormalities [7], male gender [32-34], alcohol use [35-37], snoring [26], obesity [38] (especially of the upper body), and a neck circumference of more than 17 inch in men or 16 inch in

women [39, 40]. Treatment of SAS can range from conservative methods such as oral appliances [41], to moderate intervention such as continuous positive airway pressure (CPAP) [42], to more radical approaches, including surgical removal of anatomic obstructions [43]. Since SAS is treatable, early recognition of the symptoms of this sleep disorder is very important.

II. METHODOLOGY

Data Collection

The data used in this study are obtained at Dicle University, Sleep Laboratory, Faculty of Medicine in Diyarbakir. In this study, two continuous polysomnographic (PSG) recordings have been obtained simultaneously with the channels connected as follows: Nasal airflow through a thermometer and oxygen saturation SaO_2 using a fingertip pulse oximeter. The airflow signals are digitized as 256 samples per second and SaO_2 signals are digitized as 1 sample per second. In this study 160 recordings from 10 subjects were selected for the database. The number of recordings per subject varied between 9 to 20 segments, depending on signal quality. Each recording segment is 100 seconds long. All PSG recordings were evaluated by a certified sleep specialist manually.

The overall structure of the proposed system is illustrated in Figure 1.

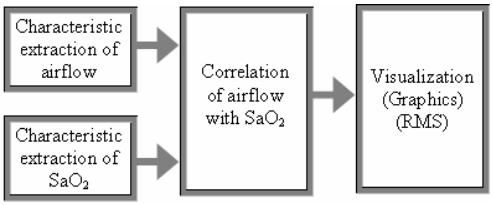


Figure 1. System overview.

Airflow Signal Processing

From the continuous PSG recordings, the airflow signals are digitized as 256 samples in 1-second frame. Most of the airflow signal power content falls between 0.1Hz and 1Hz [44]. Therefore, band pass filtering with the cutoff frequencies of 0.05 and 3Hz is performed at first to reduce the high frequency noise and the baseline drifts. From the filtered signal, the average $A_{airflow}$ and standard deviation $\sigma_{airflow}$ are calculated for each consecutive 1-second frame as follows:

$$A_{airflow}(m) = \frac{1}{M} \sum_{j=m*M}^{m*M+M-1} abs(X_{airflow}(j)) \quad (1)$$

$$\sigma_{airflow}(m) = \sqrt{\frac{1}{M-1} \sum_{j=m*M}^{m*M+M-1} (X_{airflow}(j) - A_{airflow}(m))^2} \quad (2)$$

here M is the number of samples in a 1-second frame.

The airflow signal is specific to the applied sensor and can change during long-term measurements because of sensor and patient movements. Therefore, adaptive signal normalization should be employed before features are fed into the correlation. The normalization factor is determined by,

$$F_{norm}(m) = \min\{(1-\eta)F_{norm}(m-1) + \eta.A_{airflow}(m), F_{\lim}\} \quad (3)$$

where η is the forgetting factor and set to 0.05. $F_{\lim}$ is the limit value specific to the sensor, since very small values can be the result of sensor failure. Then the normalized features are obtained by dividing each value with the normalization factor:

$$N_{airflow}(m) = \frac{X_{airflow}(m)}{F_{norm}(m)} \quad (4)$$

SaO_2 Signal Processing

Clinical experience indicates that an apneic event is associated with a decrease in the SaO_2 signal (desaturation) as well as a compensatory hyperventilatory response (resaturation) causing a rapid increase in this signal.

The tanh-normalization introduced by Hampel et al. [45] is robust and highly efficient. The normalization is given by

$$N_{SaO_2}(m) = \tanh\left(\frac{1}{T_{SaO_2}} X_{SaO_2}(m)\right) \quad (5)$$

where T_{SaO_2} is set to 4% [46].

Correlation of SaO_2 with Airflow

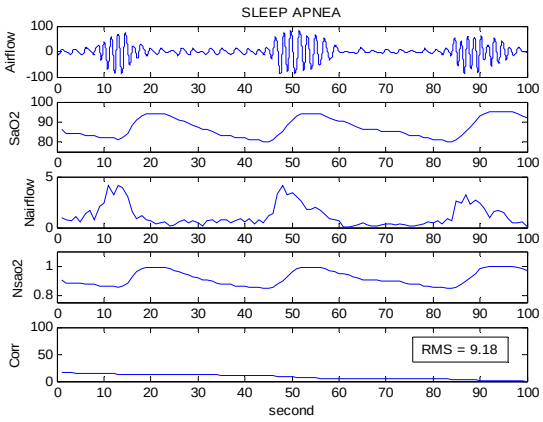
The correlation between airflow and SaO_2 is calculated with the following formula:

$$C(m) = \sum_{j=0}^{n} N_{SaO_2}(j).N_{airflow}(j+m) \quad (6)$$

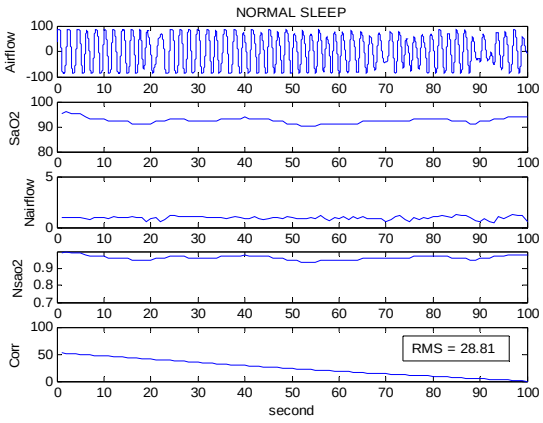
where m = 1,…,100.

III EXPERIMENTAL STUDY

In this study 7-hour night sleeping for ten patients have recorded. The data divided in 100-second segmens for each patient. For each segment, the apneic events and normal events are seperated by an expert manually.

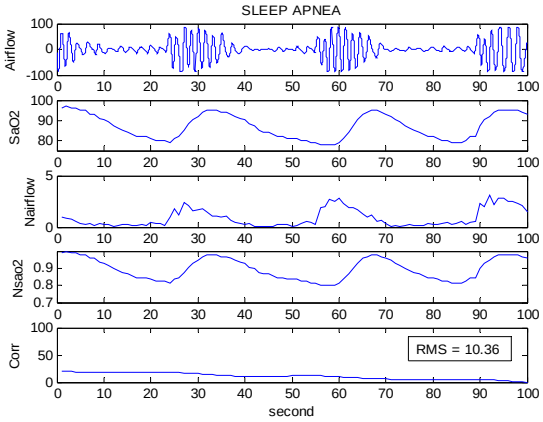


(a)

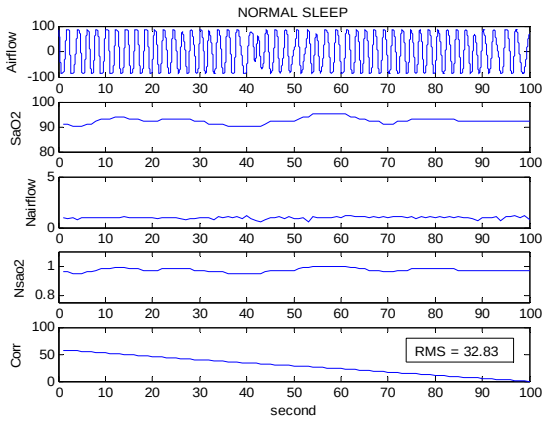


(b)

Figure 2. Steps involved in the processing procedure for a person with (a) apneic sleep and (b) normal sleep.



(a)



(b)

Figure 3. Steps involved in the processing procedure for a person with (a) apneic sleep and (b) normal sleep.

To take a close look at the overall processing procedure, Figure 2(a)-(b) and Figure 3(a)-(b) show a 100 seconds long segments from a PSG recording for sleep apnea and normal sleep. For each figure, the panels are organized as follows (from top to bottom): The original airflow signal, the original SaO_2 signal, normalized airflow averages, normalized saturation level and the correlation of airflow and SaO_2. The results of 30 segments are shown in Table 1.

DATA	EVENT	COR.(RMS)
1	Apnea	7.68
2	Apnea	8.60
3	Apnea	7.95
4	Apnea	9.25
5	Apnea	10.54
6	Apnea	6.76
7	Apnea	8.56
8	Apnea	7.12
9	Apnea	9.05
10	Apnea	9.70
11	Apnea	10.36
12	Apnea	8.21
13	Apnea	9.18
14	Apnea	7.44
15	Apnea	6.98
16	Normal	32.83
17	Normal	30.15
18	Normal	28.80
19	Normal	34.54
20	Normal	33.85
21	Normal	29.85
22	Normal	31.70
23	Normal	28.90
24	Normal	30.45
25	Normal	28.14
26	Normal	32.78

27	Normal	33.50
28	Normal	30.71
29	Normal	29.74
30	Normal	28.13

Table 1. Test results for 30 data segments

In this study, 160 data segments were tested. In Table 1, the test results for only 30 data segments are shown. The mean value and standart deviation for all 160 tested results are shown in Table 2.

Event	Total Number of Test	Mean	Standart Deviation
Sleep Apnea	82	8.56	1.27
Normal Sleep	78	30.85	2.23

Table 2. The mean and standart deviation for 82 sleep apnea and 78 normal sleep data segments.

IV. CONCLUSION

In this study, it is shown that there is a high correlation between the airflow and oxygen saturation. In sleep apnea events the correlation is low, whereas in normal sleep it is high. We have tested 160 data segments (82 apneic and 78 normal events). The results present that in sleep apnea the mean and standart deviation are 8.56 and 1.27, respectively while in normal sleep the mean and standart deviation are 30.85 and 2.23, respectively.

For sleep apnea diagnosis, many attempts adopting various signals other than airflow and SaO_2 are also performed. Those signals include snore [47], PWTT (pulse wave transit time) [48], EEG [49], and ECG [50]. Our future research will focus on the incorporation of some of those signals, which may result in a more robust detection system. In the future studies we also aim to support a software programme to the CPAP device so that it will be able to apply pressure automatically.

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