The Apnea-ECG Database

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Abstract

Sleep apnea is a sleep disorder with a high prevalence in the adult male population. Sleep apnea is regarded as an independent risk factor for cardiovascular sequelae such as ischemic heart attacks and stroke. The diagnosis of sleep apnea requires polysomnographic studies in sleep laboratories with expensive equipment and attending personnel. Sleep apnea can be treated effectively using nasal ventilation therapy (nCPAP). Early recognition and selection of patients with sleep related breathing disorders is an important task. Although it has been suggested that this can be done on the basis of the ECG, careful quantitative studies of the accuracy of such techniques are needed. An annotated database with 70 nighttime ECG recordings has been created to support such studies. The annotations were based on visual scoring of disordered breathing during sleep.

1. Introduction

Sleep related breathing disorders have a high prevalence in the adult population. Epidemiological studies indicate a high prevalence of 4% in males and 2% in females in the general population [1]. Obstructive sleep apnea, the most common of the different types of sleep-related breathing disorders, is characterized by repetitive cessations of respiratory flow during sleep.

1.1. Pathophysiology of sleep apnea

The cessations of respiratory flow occur due to a collapse of the upper airway at the level of the oropharynx. During normal inspiration the diaphragm produces negative intrathoracic pressures which in consequence inflate the lungs with air. Effective inspiration requires the upper airway to be kept open actively. In patients with obstructive apnea, there is an imbalance between the neural activation of the diaphragm and the upper airway muscles. Thus the negative intrathoracic pressure during inspiration results not in an effective airflow, but in a collapse of the upper airway. This continues until the lack of oxygen and the increase of CO_2 causes a central nervous activation, called arousal. The activation reestablishes respiration without reaching the level of conscious wakefulness, thus not being perceived by the patient. The arousal inhibits the process of falling asleep in order to reestablish the coordination of respiration for a few breaths. Then the same mechanism is repeated for the next apnea. Up to 600 apneas are recorded per night in patients with severe sleep apnea. On average, the apneas last for 40 seconds followed by a few breaths lasting for 20 seconds. During REM (rapid eye movement) sleep, when chemosensitivity is lower, apneas may last for 120 seconds.

Each apnea is accompanied by a drop in oxygen saturation. In most patients a drop of heart rate is also observed during each apnea followed by an increase of heart rate near the end of the apnea. This increase peaks during the few breaths after the apnea. This cyclic behavior of heart rate has been called cyclical variation of heart rate (CVHR) and is recognized as being specific for sleep apnea [2,3].

In parallel, sympathetic tone (recorded as sympathetic neural activity) increases during each apnea, and peaks during the few breaths following each apnea. Sympathetic tone rapidly decreases to the lowest tone which would be normal for sleep during the few breaths. In parallel with the increase of sympathetic tone, arterial blood pressure increases. Through these apnea-associated short-term cardiovascular consequences, sleep apnea appears to be an independent risk factor for arterial hypertension, and for cardiovascular sequelae such as ischemic heart attacks and stroke.

If the collapse of the upper airway is not complete, and some airflow is still recorded, the respiratory events are called hypopneas. The effect of hypopneas is the same as the effect of apneas but less pronounced (arousal, desaturation, heart rate and blood pressure variations).

The frequent arousals that terminate apneas lead to sleep fragmentation. Sleep fragmentation is characterized by disruption or loss of deep and REM sleep. Sleep loses its restorative function on physical and mental performance. Patients suffering from sleep apnea report excessive daytime drowsiness and non-restorative sleep even when the sleep period is prolonged. Bed partners may report irregular snoring and pauses in breathing during sleep. Frequent findings are obesity, hypertension, and arrhythmia.

The therapy of choice is nasal continuous positive airway

pressure (nCPAP). Room air is delivered via a nasal mask to the upper airway with a pressure of 5 to 15 cm H_2O. This pneumatically increases the pressure in the upper airway and prevents a collapse during inspiration. The nCPAP has to be used every night, with the pressure determined in a pressure titration sleep study.

1.2. Diagnosis of sleep apnea

Early recognition of sleep apnea can be done using ambulatory recording methods such as long-term ECG, long-term blood pressure and dedicated recording systems. Dedicated recording systems for the recognition of sleep-related breathing disorders record respiratory flow or movement, oxygen saturation, heart rate, body movements or body position, and snoring.

The final diagnosis of sleep apnea is done in a sleep laboratory using cardiorespiratory polysomnography [4]. This procedure requires the recording of EEG, EOG, and EMG to determine the sleep stages, oronasal airflow, chest wall and abdominal wall movements for respiratory effort, oxygen saturation to monitor the effect of respiration, and ECG for heart rate monitoring and rough arrhythmia detection. Sometimes non-invasive continuous blood pressure is recorded.

Respiratory effort can be quantified by intrathoracic pressure recording using an esophageal balloon. Airflow quantification requires a pneumotachograph with a full-face mask. Since both techniques are somewhat invasive and disturb sleep, they are not often used in sleep studies, which more often rely on indirect measurements (inductance plethysmography to track respiratory effort, and oronasal thermistors to track airflow).

The evaluation of the sleep recordings is done by visual inspection of the long-term recordings, usually with the help of computer based systems. The results of the visual analysis are a detailed description of sleep. Each 30-second epoch of the recording is classified as being awake, sleep stage 1 to 4 or REM sleep.

In addition all apneas and hypopneas are counted (fig. 1). In order to count an apnea or hypopnea, the amplitude of breathing must decrease from baseline by more than 50% for at least 10 seconds [4]. The mean duration of the apneas is estimated, and the longest events are noted.

Oxygen saturation is evaluated in terms of percent of time spent below 70%, 80% and 90% of oxygen saturation. In addition, the mean value of oxygen saturation, the total number of desaturations (more than 3%), and the lowest oxygen value are reported. To summarize the findings, an apnea index (AI), a hypopnea index (HI), and an apnea-hypopnea index (AHI) are reported, as are the number of events per hour of sleep.

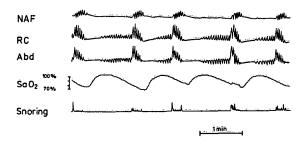The clinical diagnosis of a patient suffering from sleep

Figure 1. A sequence of four apneas is displayed. The cessation of oronasal airflow (NAF) can be observed in the top trace. Below are the traces of ribcage (RC) and abdominal (Abd) respiratory movements. Oxygen saturation (SaO_2) was recorded using a fingertip pulse oximeter (causing the delay). Snoring is quantified by the total volume of a microphone fixed at the larynx of the patient.

apnea is based on the AHI. An AHI up to 5 is regarded as normal; an AHI of 5 to 15 events per hour, as mild obstructive sleep apnea-hypopnea syndrome (OSAHS); an AHI of 15 to 30 events per hour, as moderate OSAHS; and an AHI above 30 events per hour, as severe OSAHS [4].

1.3. Computer-based apnea scoring

Computer-based systems offer automatic evaluation of apneas by analyzing the respiratory signals. In an evaluation of one such system, Taha [5] reported that if apneas and hypopneas are scored separately, the system's sensitivity for apneas was 73.6% with a specificity of 90.8%, and for hypopneas the sensitivity was 84.1% with a specificity of 86.1%. If apneas and hypopneas were taken together, sensitivity increased to 93.1%. This analysis was based on the sum of respiratory movement recording and oxygen saturation evaluation. The validation was done against the total number of apneas and hypopneas.

1.4. Limits of diagnosis

Because airflow measurement and respiratory movement recording in most sleep studies are not perfect, criteria for distinguishing apneas and hypopneas remain imperfect. Taha [5] used a decrease of respiratory movement to 20% or less of normal amplitude for at least three breaths. But what is "normal?" What minor respiratory amplitude changes may be neglected while the event is still scored as "apnea?" Each single apnea must be longer than 10 seconds in order to be counted. But when did the last expiration end? This explains the variability when scoring apneas. Variability can be low within one sleep laboratory but can be large when comparing different sleep laboratories. No published
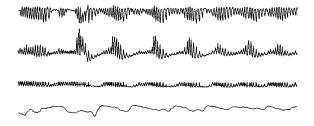
Figure 2. The transition from apnea to hypopnea to partial upper airway obstruction without severe oxygen desaturations illustrates the difficulty in scoring.

studies have compared scoring by different sleep laboratories. In general any automatic procedure can only be as good as inter-scorer variability is.

Upper airway obstruction that causes a limited airflow and in consequence also leads to arousals, sleep fragmentation and heart rate changes may also be of clinical significance. The clinical significance of partial upper airway obstruction is not proven and the criteria for discriminating between this event and hypopnea are not well defined (fig. 2). Currently apneas and hypopneas are summarized as sleep related breathing disorders, in line with the definitions for OSAHS [4].

2. ECG data in sleep apnea

Heart rate, and other characteristics of the ECG, vary in characteristic ways in parallel with sleep related breathing disorders [2]. Previous work made use of these cyclical variations of heart rate but did not establish valid algorithms to quantify sleep related breathing disorders based on heart rate alone [3, 6]. It is obvious that an evaluation of heart rate cannot produce an apnea index or an hypopnea index. Both values are defined by the evaluation of airflow, respiratory effort and oxygen saturation. The evaluation of ECG can give an estimate of disturbed breathing during the night which should correspond to the results obtained by standardized apnea scoring.

3. Subjects and recordings

A total of 70 sleep recordings with good ECG and respiratory signals were selected for inclusion in the Apnea-ECG Database. The sleep recordings were selected from two earlier studies where the ECG was not the focus of the research. The first study, undertaken between 1993 and 1995, investigated the effect of obstructive sleep apnea on arterial blood pressure, in subjects with moderate and severe sleep apnea. The AHI varied between 5 and 75 respiratory

events per hour. All subjects were recorded for two consecutive nights, and then after an interval of four weeks, for two more consecutive nights. The ECGs were digitized at 100 Hz. Twenty-seven recordings from nine subjects in this study were selected for the database. The number of recordings per subject varied between one and four, depending on signal quality.

During 1998 and 1999, the second study was undertaken in order to create a normative set of sleep recordings. In this study, the focus of the research was a multiple channel EEG recording. The study was performed on healthy volunteers and selected patients with sleep apnea. The AHI in this set of apnea patients varied between 14 and 82 events per hour. All subjects were recorded for two consecutive nights. The ECGs were digitized at 200 Hz, and later decimated to 100 Hz for consistency with the data from the first study. Forty-three recordings from 23 subjects in the second study were selected for the database; for each subject, no more than two recordings were selected.

3.1. Scoring of time spent in disordered breathing

The initial scoring of apneas and hypopneas in the data was done according to standard criteria. The number of apneas and hypopneas were determined within the evaluation of the original studies. These values were reported when reference is made to AI and AHI here in order to characterize the data.

The database is compiled to detect sleep related breathing disorders based on a single channel ECG recording. For this specific aim all polysomnographic recordings were scored again by one expert (TP) in a different way. This new scoring did not differentiate between apnea and hypopnea events. The result of the scoring were markings for the beginning and the end of episodes of disordered breathing. The disordered breathing may contain one single apnea or hypopnea or may contain a longer sequence of apneas and hypopneas. The markings were mapped to time with a resolution of one minute. The final result of the scoring was a binary information for each minute of the recording being coded as either "normal breathing" (N) or "disordered breathing" (A). The total number of minutes spent in apneas or hypopneas was determined for each recording.

4. Groups of subjects

The duration of the ECG recordings for the 70 recordings varied between 401 and 578 minutes. The time spent in normal breathing varied between 11 and 535 minutes. The time spent in disordered breathing varied between 0 and 534 minutes. Based on the number of minutes spent with disordered breathing, we defined three groups of recordings.

Recordings having fewer than 5 minutes of disordered breathing were put in the normal (control, or class C) group. Twenty recordings met this criterion, and each had between 0 and 3 minutes of disordered breathing. The group consisted of six male and five female subjects with a mean age of 33 years (27-42 years).

The group of apnea (class A) recordings was defined as having 100 or more minutes with disordered breathing. Forty recordings were in this category, each with between 100 and 534 minutes of disordered breathing. The subjects of the class A recordings were fifteen men and one woman, with a mean age of 50 years (29-63 years).

In between these two groups, we defined the "borderline apnea" (class B) group with some apneas of uncertain importance. This group contained 10 recordings, each with 10 to 96 minutes of disordered breathing. The subjects were four men and one woman, with a mean age of 46 years (39-53 years).

When we looked in the records of the recordings rated as "borderline apnea", we found that they originated from both healthy volunteers and apnea patients. One healthy subject turned out to have some apneas in one recording night and another healthy subject had some apneas in both recording nights. Those three nights were taken as "borderline apnea" recordings, as were recordings from three subjects in the first study in which relatively few apneas were observed.

5. Discussion

The scoring used for the database of "time spent in disordered breathing" instead of apneas and hypopneas avoided the difficulty of distinguishing apneas and hypopneas, focusing rather on the distinction between normal breathing and disturbed breathing. As the respiratory signals in our sleep studies originate from thermistor-based airflow recordings and inductance plethysmography-based respiratory effort recordings, the scoring is subjective.

When disordered breathing in sleep is determined based on a surrogate parameter such as the ECG and heart rate, it is more appropriate to calculate minutes spent with disordered breathing than the total number of respiratory events expressed as apnea-hypopnea index.

All data have been made freely available via PhysioNet [7], initially to support the Computers in Cardiology Challenge 2000 [8]. Thirty-five of the ECG recordings were provided together with apnea annotations, and eight of these were accompanied by respiration and oxygen saturation signals. For the other 35 recordings, apnea annotations were withheld for the duration of the challenge.

Acknowledgements

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