

A novel method to precisely detect apnea and hypopnea events by airflow and oximetry signals



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ABSTRACT

Sleep apnea hypopnea syndrome (SAHS) affects people's quality of life. The apnea hypopnea index (AHI) is the key indicator for diagnosing SAHS. The determination of the AHI is based on accurate detection of apnea and hypopnea events. This paper provides a novel method to detect apnea and hypopnea events based on the respiratory nasal airflow signal and the oximetry signal. The method uses sliding window and short time slice methods to eliminate systematic and sporadic noise of the airflow signal for improving the detection precision. Using this algorithm, the sleep data of 30 subjects from the Huaxi Sleep Center of Sichuan University (HSCSU) and the Teaching Hospital of Chengdu University of Traditional Chinese Medicine (THCUTCM) were auto-analyzed for detecting the apnea and hypopnea events. The total predicted apnea and hypopnea events were 8470. By manual investigation, the sensitivity and positive predictive value (PPV) of detecting apnea and hypopnea events were 97.6% and 95.7%, respectively. The sleep data of 28 subjects from HSCSU were auto-diagnosed SAHS according to the AHI. The sensitivity and PPV were 92.3% and 92.3%, respectively. This is an effective and precise method to diagnose SAHS. It can fit the home care SAHS screener.

1. Introduction

Approximately 2–4% of the adult population worldwide suffered from sleep apnea hypopnea syndrome (SAHS) in 1993 [1]. The estimated prevalence rates of the SAHS had increased over the last 2 decades since 1993; it relatively was increased between 14% and 55% depending on the subgroup in 2013 [2]. SAHS is related to cardiac disease, diabetes, hypertension and many other major diseases [3–12]; it is a risk factor for sleep stroke also [13]. Some studies indicate that there is a relationship between obstructive sleep apnea syndrome (OSAS) and cancer mortality [14]. In general, OSAS is accompanied by daytime sleepiness and tiredness; thus, this disease is associated with the incidence of traffic accidents [15–18].

A recent study has pointed out there is uncertainty about the accuracy or clinical utility of all potential screening tools [19]; however, so far the gold standard for SAHS disease diagnosis is polysomnography (PSG). Except to PSG diagnosis, more simple equipment is used to screen the disease based on different methods; moreover, the home care device for diagnosing OSAS is meaningful [20]. Some methods have depended on a single oxygen signal [21–23]; some algorithms have relied on a single airflow signal [24–26]. An algorithm depended on

nighttime snoring [27]. Another method based on a nasal pressure signal [28]. Recently, more studies have focused on detecting respiratory events from the ECG signal [29–32]. Home care OSAS screening is becoming increasingly important. However, more effective, robust and precise algorithms are the focus of current efforts in the design of an OSAS screener [33].

The algorithm in this paper is based on the 2012 American Academy of Sleep Medicine (AASM) criteria [34]. It scores the respiratory events using nasal airflow and oximetry signals. It improves the scoring accuracy by eliminating the impact of systematic and sporadic changes of the baseline amplitude of the nasal airflow signal.

In the algorithm, the systematic and sporadic changes of the nasal airflow baseline are eliminated by a sliding window and a series of short time slices of the respiratory signal. At first, the whole recorded airflow signal is divided into a series of short time slices. Then, a width limited sliding window slides over all of the short time slices for analyzing the time slice state. Next, unclassified respiratory events are obtained according to the adjacent time slice state. Then, the unclassified events were classified as apnea or hypopnea events by the Bayesian criterion. Finally, the hypopnea events must be filtered by the oxygen desaturation event of the corresponding period.

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All original sleep data used by the algorithm were recorded by a SR20C portable sleep screener (SR20C) manufactured by Chengdu Easyhealth Technology Co., Ltd, Chengdu city, Sichuan province of China. The SR20C simultaneously records seven types of signals, including nasal airflow, thoracic and abdominal respiratory motion, pulse oximetry, heart rate, snores and sleep posture. The nasal airflow and oximetry signals among the seven signals were used by the algorithm for analyzing the respiratory events. The hardware parameters of the nasal airflow are as follows: sample rate is 50 Hz; filter band is from 0.05 Hz to 5 Hz; and ADC resolution is 12 bits. The pulse oximetry probe is a digital SpO₂ probe. The sample rate is 1 Hz, with a range from 40% to 99% and a resolution of 1%. SR20C itself uses the algorithm.

Two groups of analyzed sleep datasets recoded by SR20C came from the Huaxi Sleep Center of Sichuan University (HSCSU) and the Teaching Hospital of Chengdu University of Traditional Chinese Medicine (THCUTCM). Among them, 28 sleep datasets came from HSCSU and 30 sleep datasets came from THCUTCM. The 58 sleep datasets achieved patient consent for the study. The demographics of 58 sleep data see Table 1. The 28 sleep datasets were selected randomly from two groups of sleep datasets from HSCSU and THCUTCM were taken as the training data used to get the priori and conditional probability for a Bayesian classifier. The remaining 30 sleep datasets from HSCSU and THCUTCM were used as testing data to verify the effectiveness and accuracy of the algorithm for classifying the apnea and hypopnea events. The 30 sleep datasets were automatically analyzed by the algorithm; a total of 8470 respiratory events were detected. The all apnea and hypopnea events auto analyzed by the algorithm were validated independently by two trained technician according to 2012 AASM criteria. The specificity and PPV of respiratory event detection were 97.6% and 95.7%, respectively.

PSG is taken as a diagnosing gold standard. The 28 sleep datasets from HSCSU were simultaneously recorded by the Alice5 PSG that was manufactured by Philips Respironics Company. The Alice5 PSG equipment belonged to HSCSU. The AHI of 28 simultaneously recorded datasets were independently auto analyzed by the Alice5 PSG and the algorithm. The correlation coefficient of the two groups of AHI results is 0.94.

This paper is divided into six sections. The first section contains the introduction. The second section analyzes the factors that affect the determination of respiratory events. The third section introduces the main algorithm. The fourth section provides the results. The fifth section presents the discussion. The last section presents the conclusions.

2. Factors that affect determination of respiratory events

There are many factors that affect the determination of the respiratory event; for example, the quality of the sampling signal, the judging criteria, and the dynamic changes in the respiratory amplitude and morphology.

2.1. Criteria for determining apnea and hypopnea events

The apnea and hypopnea events are scored in this paper according to the 2012 AASM criteria. Scoring a respiratory event in adults as apnea requires meeting two conditions: 1) there is a decrease in the peak signal excursion by $\geq 90\%$ of the pre-event baseline; and 2) the duration of the $\geq 90\%$ drop is ≥ 10 s. The respiratory event is scored as hypopnea if all of the following were met: 1) the peak signal excursions decrease by $\geq 30\%$ of the pre-event baseline; 2) the duration of the $\geq 30\%$ decrease in signal excursions is ≥ 10 s; and 3) there is $\geq 3\%$ oxygen desaturation from the pre-event baseline or the event is associated with an arousal [34].

2.2. Systemic change in baseline amplitude

The baseline amplitude is the respiratory amplitude in the normal respiratory condition, the basis for determining the respiratory event.

Table 1

The demographics of 58 sleep data came from HSCSU and THCUTCM.

Subject	Male	Female	Average Age	Range of age
Number	41	17	45 \pm 15	19–75
Proportion	70.7%	29.3%		

The baseline amplitude is not always stable. A systemic change typically occurs based on the following description. In a long period, such as 10 min or more, the baseline amplitude remained stable with subsequent quick increases or decreases greater than 50% relative to the previous baseline amplitude and then remains stable for another long period. The baseline amplitude may be changed by more than 50%; however, the amplitude in the next long period continuously maintains the relative stability. Moreover, it does not accompany oxygen desaturation events; thus, it is also considered to be normal breathing. The systemic change in the baseline amplitude may indicate a natural respiratory slowdown during sleep or it may indicate that the nasal catheter position changed or even fell off (see Fig. 1).

The systemic change in the baseline amplitude may be classified into reversible and irreversible changes. Reversible systemic changes include a decrease or increase in the baseline amplitude. These changes may be caused by a change in the nasal airflow catheter position. The baseline amplitude increases when the catheter is close to the nostrils. However, the amplitude decreases when the catheter is away from the nostrils. If the nasal airflow catheter falls off or the recording device is abnormal, the amplitude change is irreversible. In this condition, the respiratory amplitude may be close to zero; the process of analyzing the respiratory event is typically stopped when there is an irreversible change in this algorithm.

2.3. Sporadic change in respiratory amplitude

In addition to systemic changes, there are many factors that may cause sporadic changes in the respiratory amplitude, e.g., the user turned over or coughed. This sporadic change may cause a significant change in the respiratory amplitude in a short duration (see Fig. 2).

3. Algorithm

The algorithm is referred to as short time slice event state (STSES) detection, and the state flow of short time slice (SFSTS) generates the respiratory events algorithm. The purpose of the algorithm is to eliminate the influence of systemic or sporadic changes in the respiratory amplitude to more accurately identify respiratory events.

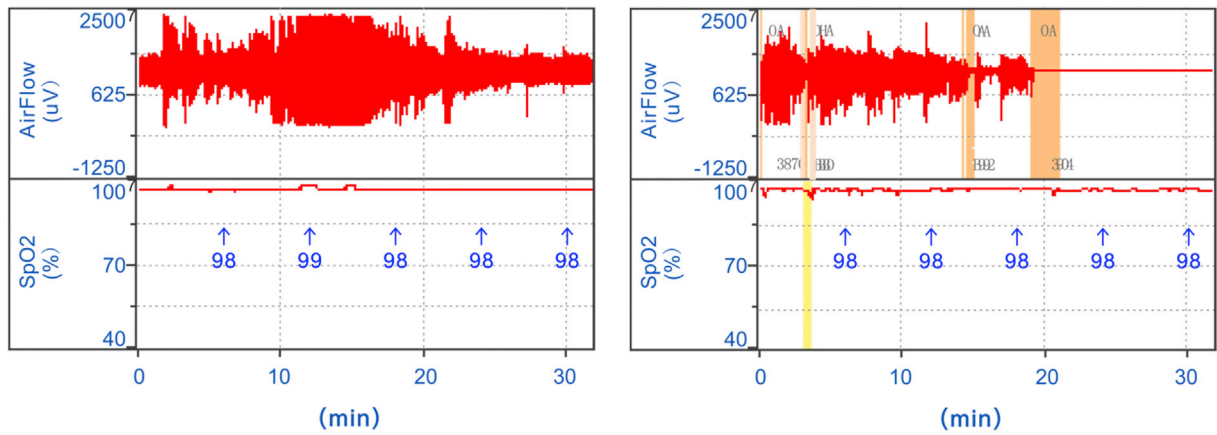
The core idea of the algorithm is to dynamically track and decrease the influence of systematic changes in baseline amplitude using a sliding window and filter out the influence of sporadic change in the respiratory amplitude using the short time slice method.

According to the idea, the algorithm mainly contains two basic processes:

- 1) Converting the original respiratory signal into the SFSTS
- 2) Generating different respiratory events from the SFSTS

In the first step, a sliding window including many short time slices slides over the whole original respiratory signal; then, all short time slices are converted into the SFSTS. There are three basic states: normal, apnea and hypopnea states. In the second step, the multi adjacent STSES are merged into a respiratory event according to the 2012 AASM criteria until the whole SFSTS is converted into respiratory events. The process is illustrated in Fig. 3.

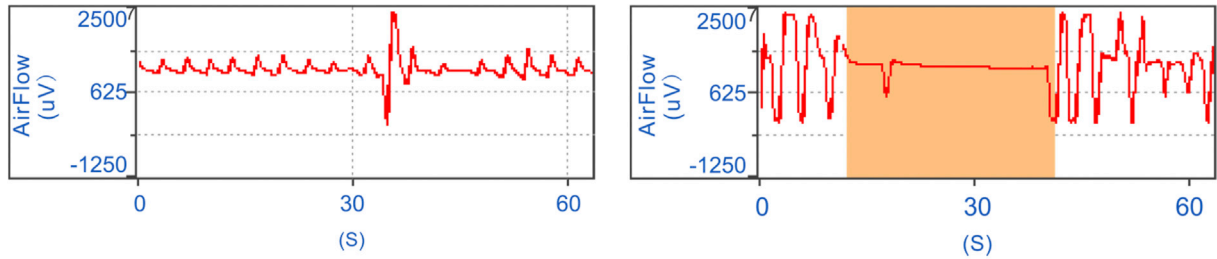
In Fig. 3, N is the normal state, A is the apnea state, and H is the hypopnea state.



(a) Reversible systemic changes in the respiratory amplitude may be caused by a change in the nasal airflow catheter position

(b) Irreversible systemic changes in the respiratory amplitude may be caused by the nasal airflow catheter falling off or an abnormal recording device

Fig. 1. Systemic changes in respiratory amplitude (channel 1 is a compressed airflow signal, and channel 2 is the corresponding SPO2 signal).



(a) Sporadic change in respiratory amplitude in the normal respiratory signal

(b) Sporadic change in respiratory amplitude in a respiratory event signal

Fig. 2. Sporadic changes in respiratory amplitude.

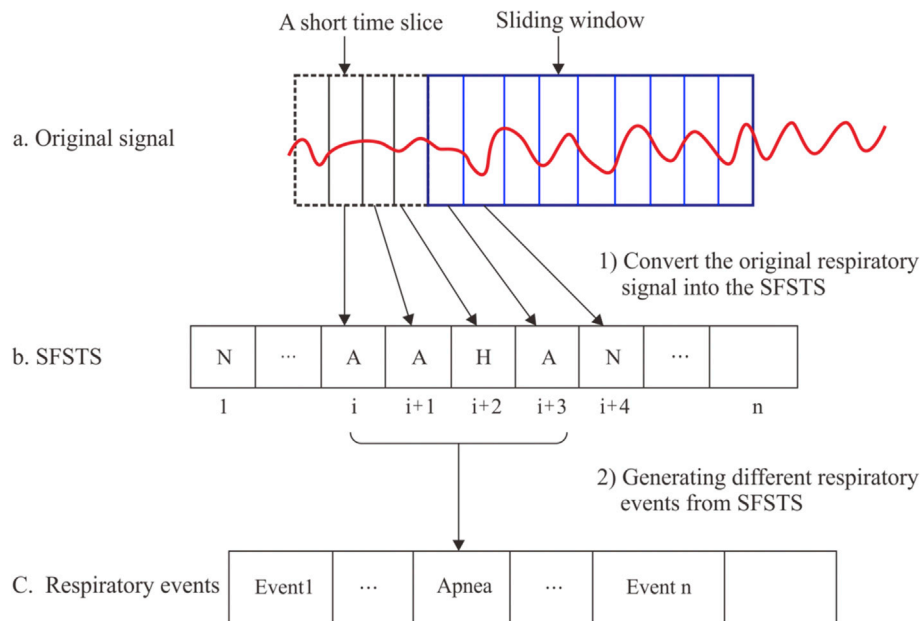


Fig. 3. Illustration of the main process for determining respiratory events.

3.1. Converting the original respiratory signal into the SFSTS

The first step in converting the original respiratory signal into the SFSTS has six sub processes (see Fig. 4).

In this process, there are some problems that need to be solved. The

first problem is how to determine the duration of the short time slice. The second problem is how to calculate the amplitude of the short time slice. The third problem is how to calculate the baseline amplitude of a sliding window. The last problem is how to convert the amplitude of the short time slice into the corresponding state.

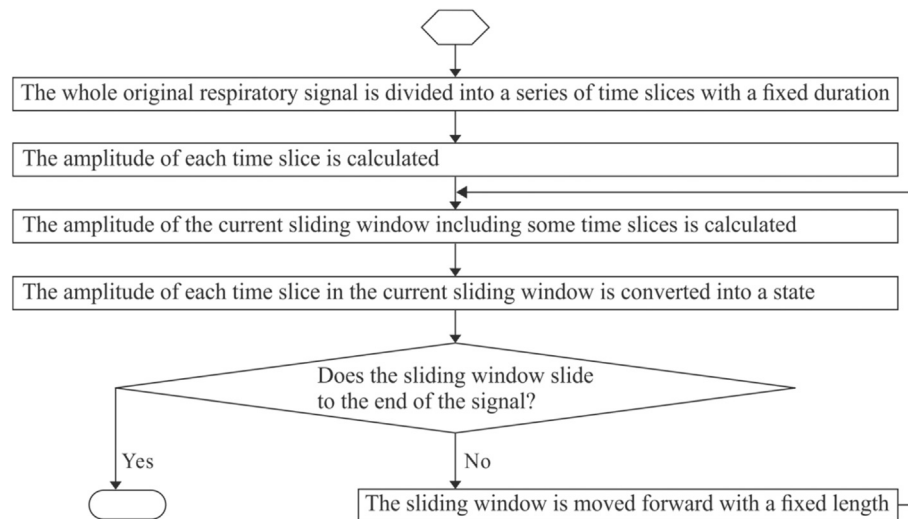


Fig. 4. Process of converting the original respiratory signal into the SFSTS.

3.1.1. Determining the duration of the short time slice

In this algorithm, the fixed short time slice (FSTS) is the basis of the following analysis. The principle of selecting the duration of the FSTS is that each time slice only contains a respiratory wave.

There are three conditions to discuss regarding the selection of the duration.

1) The duration of the FSTS is greater than the current respiratory period

When the selected duration of the FSTS is greater than the current respiratory cycle, one to n respiratory waves exist in a FSTS. The respiratory amplitude may be obtained from a FSTS. However, there are two problems in the condition. The first problem is when there is respiratory fluctuation; the obtained respiratory amplitude may be greater than the baseline amplitude. Another problem is that the long time slice will cause a fuzzy boundary of the obtained respiratory event because the long time slice decreases the time resolution.

2) The duration of the FSTS is equal to the current respiratory period

In this case, the FSTS contains only one respiratory wave; the peak-to-trough amplitude (PTA) of the FSTS is equal to the baseline amplitude. However, this situation is an ideal state. The duration of the FSTS being equal to the respiratory cycle does not exist because of the fluctuation of the respiratory period during sleep [35].

3) The duration of the FSTS is between one-half and one current respiratory period

If the respiratory wave is considered to be the sine wave, when the duration of the FSTS is more than half and less than one respiratory cycle, the PTA of the normal respiratory wave would be detected in multiple continuous FSTSs. Moreover, the amplitude of FSTS is not easily affected by the abnormal respiratory wave; it has a higher time resolution to detect the respiratory event boundary and is advantageous in obtaining the event duration. Therefore, the algorithm chooses the shorter time slice.

Previous knowledge indicates that the normal human breath cycle in sleep is between approximately 3 and 5 s [35] [36]; the typical value is approximately 4 s. Therefore, the duration of the FSTS is chosen to be between 2 and 5 s.

The calculation of the real-time respiratory cycle is easily affected by the respiratory fluctuation and the irregular morphology of the respiratory wave. To simplify the algorithm and avoid an inaccurate respiratory

cycle, a minimum duration of 2 s was considered to be the duration of the FSTS in this algorithm.

3.1.2. Calculating the amplitude of the short time slice

The PTA value of the FSTS is considered to be its amplitude. The formula for calculating the PTA value of a FSTS is as follows:

$$V_{pt}(i) = V_{max}(i) - V_{min}(i) \quad (1)$$

where the $V_{pt}(i)$ is the PTA value of the i th FSTS, $V_{max}(i)$ is the maximum value of the i th FSTS, and $V_{min}(i)$ is the minimum value of the i th FSTS.

Because the selected duration of the FSTS is commonly shorter than the respiratory cycle, the FSTS typically does not synchronize with the respiratory cycle. Therefore, some FSTSs do not include the PTA values of a normal respiratory wave. The amplitude of these FSTSs is smaller than the baseline amplitude. However, according to anterior analysis, there are some FSTSs that must contain the PTA value of the normal respiratory wave. As long as the baseline amplitude is included in these FSTSs, it will be selected in the subsequent calculation within the sliding window.

3.1.3. Calculating the baseline amplitude of the respiratory wave in the current sliding window

Because of the systematic change in the baseline amplitude, it is impossible to obtain only one fixed baseline amplitude to determine all of the respiratory events. To eliminate the influence of the systematic change on the baseline amplitude, a sliding window is used to track and dynamically calculate the baseline amplitude.

How long is the sliding window? To obtain the correct baseline amplitude, it is ensured that at least one normal respiratory wave must exist in a sliding window. In general, there are no normal respiratory waves in the apnea and hypopnea events. Therefore, the duration of the sliding window must span the longest duration of a respiratory event. However, the longest duration of the respiratory event was not defined in the AASM criteria and no other evidence has been published regarding this problem. Hence, by pre-analyzing the duration of the respiratory events in 58 sleep datasets from HSCSU and THCUTCM, the longest duration of a respiratory event is almost 120 s. To calculate the correct baseline amplitude, the duration of the sliding window should be equal to the longest duration of the respiratory event plus some normal respiratory cycles. The normal respiratory cycle is typically approximately 4 s. If the sliding width includes four normal respiratory waves at a minimum, it is approximately 16 s. The sliding window may slide the longest

duration of the respiratory event each time. Thus, the last window has some overlap with the current window.

How is the correct baseline amplitude of a sliding window calculated? A sliding window includes many FSTs, and each time slice has its own amplitude. According to the pre-analysis, the baseline amplitude of a sliding window exists in its FSTs amplitude. The problem then becomes correctly selecting the baseline amplitude from the FSTs amplitude in a sliding window.

In a sliding window, there may be a normal respiratory wave, abnormal respiratory wave and respiratory events. A normal respiratory wave includes correct baseline amplitude, but an abnormal respiratory wave may cause a large sporadic change in the baseline amplitude. The respiratory events have smaller amplitude than the baseline amplitude.

For filtering the incorrect amplitude in the FSTs of the current sliding window, the amplitude of all FSTs in the sliding window was initially sorted. Then, the amplitude of the FSTs between low and high thresholds of the last baseline amplitude was selected. The low threshold should be below 70% of the last baseline amplitude because amplitude of less than 70% of the baseline is considered as the respiratory event according to the 2012 AASM criteria. The high threshold is below 150% of the last baseline amplitude because amplitude more than 150% of the baseline is considered to be a sporadic change as a result of noise (see Fig. 2). The arithmetic mean value of the amplitude of the selected FSTs was considered to be the preliminary baseline amplitude of the current sliding window.

The obtained preliminary baseline amplitude will be optimized. In general, the baseline amplitude is typically associated with the history; thus, the current calculated preliminary baseline amplitude was weighted with the historic baseline amplitude, and the weighted result takes the baseline amplitude of the current sliding window. The formula used to calculate the weighted baseline amplitude is as follows:

$$BA(i) = K1 \times BAH(i) + K2 \times BAC(i) \quad (2)$$

The $BA(i)$ is the baseline amplitude that is used to determine the respiratory events in the i th sliding window. The $BAH(i)$ is the historic baseline amplitude. $K1$ is the weight of the historic baseline amplitude. The $BAC(i)$ is the current baseline amplitude. $K2$ is the weight of the current baseline amplitude. $BAC(i)$ is typically a higher weight. The range of $K1$ is $[0, 0.5]$; the range of $K2$ is $(0.5-1]$, and $K1 + K2 = 1$. The $BAH(i)$ may be replaced by $BA(i-1)$. When i is equal to zero, the $BAH(i)$ is set to equal to $BAC(i)$. The baseline amplitude of the first sliding window $BA(0)$ is the mean value of the amplitude of the selected FSTs.

3.1.4. Converting the amplitude of a FSTs to a state of the FSTs

Each sliding window has baseline amplitude. Using the baseline amplitude compares the amplitude of each FSTs with the current sliding window. According to the compared result and the 2012 AASM criteria for determining the respiratory event, the amplitude of each FSTs in the current sliding window was converted into a state. There are three states: normal, apnea and hypopnea states. When the sliding window slides

across the entire respiratory signal, the whole respiratory wave becomes the SFSTs.

The state of a FSTs is not really a respiratory event because the shortest duration of the respiratory event is longer than twice the duration of the longest FSTs. In general, the states of multiple continuous FSTs are merged into a respiratory event.

When the amplitude of the FSTs is converted into states, some states could be wrong. These wrong FSTs states do not represent the true states. For example, the duration of the FSTs is shorter than the respiratory cycle; thus, the amplitudes of certain FSTs are smaller than the baseline amplitude. In this condition, even if the true state of the FSTs is normal, it may be taken as a state of a respiratory event when the amplitude of the FSTs is smaller than the threshold of the determining baseline. However, the local incorrect states do not have an impact on the result of determining a respiratory event. A respiratory event is typically a long duration decrease in the respiratory amplitude; thus, multiple continuous respiratory STSESs may be merged into a respiratory event. The discontinuous error state of the FSTs will be filtered in the subsequent process.

3.2. Generating respiratory events from the SFSTs

The second process of generating different respiratory events from the SFSTs is illustrated in Fig. 5.

3.2.1. Converting the SFSTs into the primary respiratory event

A respiratory event comprises multiple continuous correlative STSESs. Therefore, the converting process is the process of merging some STSESs to an event until the end of SFSTs.

There are two alternate processes for converting STSES to a respiratory event. The first process is the searching STSES process. The second process is generating a respiratory event process according to the correlative STSES. The second process includes three sub processes: start, growth and end. The state transformation process is illustrated in Fig. 6.

The searching STSES process indicates that the first STSES has not yet been found from the following SFSTs in the current searching process. This process is the beginning of the whole converting process. When the first STSES has been found, the process enters into the generating respiratory event process.

The generating respiratory event process indicates that some correlative STSESs have been identified and have been merged into the current generating respiratory event. When a current generating respiratory event process finishes the main converting process, it returns back to the searching STSES process again. The two processes alternately occur until the whole SFSTs has been completely processed.

The generating respiratory event process is relatively complex. It includes three sub processes: start, growth and end.

1) Start sub process

The start sub process initializes some condition of generating a

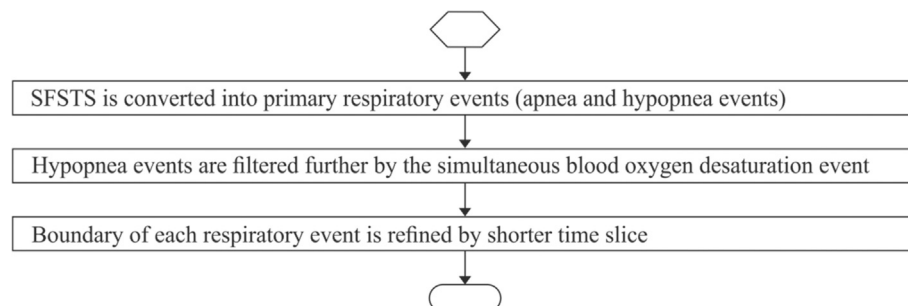


Fig. 5. Process of generating different respiratory events from the SFSTs.

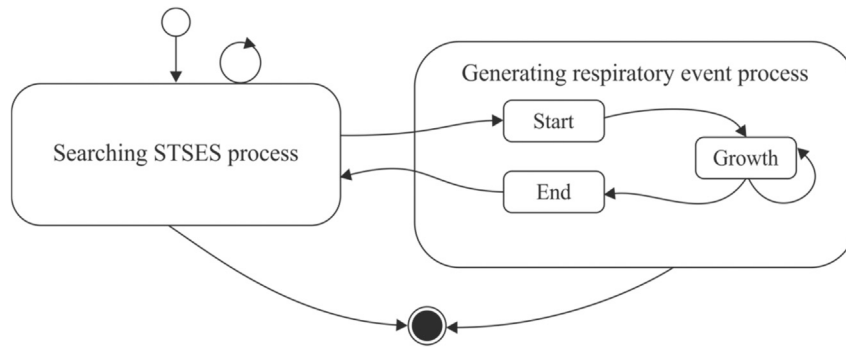


Fig. 6. State transformation process for determining a respiratory event.

respiratory event, e.g., various STSES counters, such as an apnea and hypopnea STSES counter.

2) Growth sub process

The event growth sub process is a process that extends the current generating respiratory event according to the current, historical and future short time slice state until the generating event is terminated. If the next short time slice state is the STSES, it will be merged into the current generating respiratory event.

A few of the interferences in the respiratory event were allowed according to the 2012 AASM criteria for judging a respiratory event. The normal state may be considered to be the noise in a respiratory event (see Fig. 7). If the proportion of the normal state is less than a certain limit; the growth event should continue to grow.

3) End sub process

Respiratory event growth is terminated by three situations. The first situation occurs when the duration of the current generating event exceeds the maximum limit. The second situation occurs when two non-continuous normal states occur in the current generating event. The third situation occurs when there are two continuous normal states when the event grows. In the third situation, the last two normal states of the time slice are excluded in the generated respiratory event.

When the current generating event is being terminated, the event will be discarded if the duration of the generated event is shorter than the minimum duration of respiratory event of the 2012 AASM criteria. Otherwise, the generated event will be classified as an apnea or hypopnea event according to the Bayesian criterion.

If all states in the generated event are consistent, then the event will be definitely classified as an apnea or hypopnea event. However, apnea and hypopnea states sometimes simultaneously exist in the generated respiratory event (see Fig. 7).

In the 2012 AASM criteria for judging a respiratory event, the percentage that met the amplitude decrease was required to be greater than 90%. Because the time resolution of the FSTS is low, it is difficult to

strictly satisfy the greater than 90% principle in this algorithm only according to the proportion of a type of STSES. The edge of the generated respiratory event may easily cause incorrect judging. For example, at the beginning of the respiratory amplitude decrease, the state of the whole edge may be determined to be a hypopnea state. However, if the time slice has a higher time resolution, the whole edge may be divided into two components. The anterior part is the hypopnea state because of the high amplitude; the posterior part should be an apnea state because of the low amplitude. Therefore, it is difficult to guarantee that the percentage of the single event state of a generated respiratory event is greater than 90%. Thus, the Bayesian criterion is considered to be the classifier for distinguishing apnea and hypopnea events.

According to the Bayesian criterion, the algorithm classifies the generated respiratory event to an apnea or hypopnea event based on the proportion of the apnea state in the total states composed of the unclassified respiratory event.

The event space contains two types of events: apnea and hypopnea. The Bayesian formula used to classify the unclassified respiratory event is as follows:

$$P(Ei|APr) = \frac{P(Ei) \times P(APr|Ei)}{\sum_{i=1}^N (P(Ei) \times P(APr|Ei))} \quad (3)$$

Ei represents a type of respiratory event. There are two types of respiratory events: apnea and hypopnea events. N is the classified number of the respiratory event, i.e., two. APr represents the percentage of the apnea state number relative to the total state number in a generated respiratory event. APr indicates the percentage range of the apnea state number relative to the total state number in a respiratory event, such as 50–60% or 70–80%.

P(Ei) is the priori probability of a type of respiratory event. P(APr|Ei) represents the conditional probability that the percentage range of the apnea state number relative to the total state number is within a specified range in the respiratory event Ei. P(Ei|APr) indicates the posterior probability that the Ei event is identified according to the percentage range APr of the apnea state number relative to the total state number.

The 28 sleep datasets randomly selected from the HSCSU and THCUTCM were analyzed. A total of 6379 apnea events and 11038

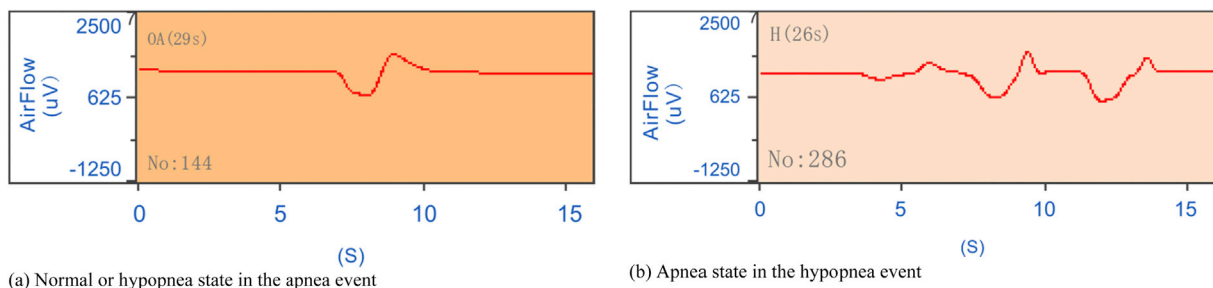


Fig. 7. Different states simultaneously exist in the generated respiratory event.

Table 2

Conditional probabilities of apnea state proportion range in apnea and hypopnea events.

Apnea state proportion range	P(Apr Apnea)	P(Apr Hypopnea)
[0%, 10%)	0	0.719
[10%, 20%)	0	0.133
[20%, 30%)	0	0.090
[30%, 40%)	0	0.014
[40%, 50%)	0	0.026
[50%, 60%)	0.079	0.017
[60%, 70%)	0.166	0
[70%, 80%)	0.091	0
[80%, 90%)	0.132	0
[90%, 100%]	0.533	0

hypopnea events were analyzed from the 28 sleep datasets. The prior probability of an apnea event P(AE) is 0.366 and the prior probability of a hypopnea event P(HE) is 0.634. The conditional probabilities of the apnea state proportion range in the different respiratory events are shown in Table 2.

In Table 2, the apnea state proportion range is apnea state percentage range in a respiratory event. P(Apr|Apnea) is the conditional probability that the percentage range of the apnea state is within a specified range in an apnea event. For example, the conditional probability is 7.9% that percentage range of apnea state between 50% and 60% in an apnea event. P(Apr|Hypopnea) is same to P(Apr|Apnea).

3.2.2. Filtering hypopnea events according to the oxygen desaturation event

The 2012 AASM criteria for determining a hypopnea event includes at least two conditions. One condition is a decrease in the respiratory amplitude. Another condition is that the oxygen desaturation event simultaneously occurs. According to the criteria, determination of a hypopnea event is required to simultaneously exhibit an accompanying 3% oxygen desaturation event or arousal. In reality, the oxygen desaturation event typically has a certain delay relative to the decrease in the respiratory amplitude [37]. Thus, the algorithm only requires that a time overlap exists between the end of the hypopnea event and the oxygen desaturation event. The generated hypopnea events that do not accompany an oxygen desaturation event should be discarded.

3.2.3. Refinement of respiratory event boundaries

The refinement of the respiratory event boundary is meaningful to obtain a more accurate duration of the respiratory events.

The method of refining an event boundary is to re-judge the state of the two boundaries of each identified respiratory event in a shorter time slice. The shorter time slices in which the state is the same as the determined event and are next to the event are merged into the event. Other shorter time slices in which the state differs from the determined event are discarded.

4. Results

4.1. Duration of FSTS impacts on analysis results

Only the duration of the FSTS was changed. The AHIs of the 30 sleep datasets from HSCSU and THUCM were analyzed, and the average values of the 30 AHIs were calculated. The results are shown in Table 3.

Table 3 indicates that the analysis results obtained in different durations of the FSTS are mildly different. The average AHI is 28.79, and the standard deviation (SD) is 1.60. Relatively, the analysis result is little

Table 3

Influence of different durations of the FSTS on the analysis results.

Duration	2 s	3 s	4 s	5s	Average	SD
AHI	31.55	27.99	27.70	27.9	28.79	1.60

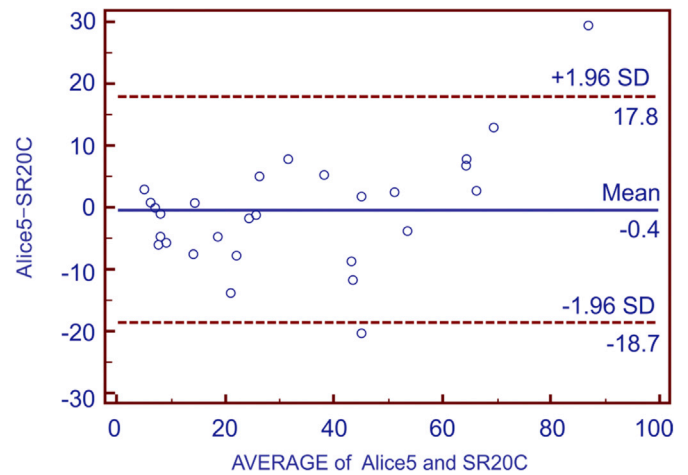


Fig. 8. Bland-Altman plots illustrating the agreement of the analysis results between the Alice5 PSG and SR20C with a mean difference $-0.4/h$.

higher by 2s duration of the FSTS because 2s duration analysis may get more respiratory events of 10s critical duration. Furthermore, taking into account that a longer duration will cause the event boundary to become fuzzy, a shorter duration of the time slice is thus recommended.

4.2. Result compared to the Alice5 PSG

There are 28 valid sleep datasets that were simultaneously recorded by the SR20C and the Alice5 PSG of HSCSU. The 28 recorded datasets from the SR20C were analyzed by the algorithm of the paper, and the 28 simultaneously recorded datasets from the Alice5 PSG were automatically analyzed by the Alice5 PSG program independently. The average AHI was 32.97 when analyzed using this algorithm. The average AHI was 32.55 when analyzed by the Alice5 PSG. The correlation coefficient of the AHI value of the two groups was 0.94. Fig. 8 presents a Bland-Altman graph for a comparison of the differences between the two groups of results. The mean difference is $-0.4/h$.

The AHI value analyzed by this algorithm is consistent with the AHI obtained by the Alice5 PSG. However, there are four factors affecting the result of the comparison. The first factor is the different original sleep dataset that was simultaneously recorded by the SR20C and the Alice5 PSG. The second factor is that the algorithm is different between that from the Alice5 PSG and this algorithm. The third factor is that a difference may exist in the total sleep time (TST) that is used to calculate the AHI. The Alice5 PSG may automatically calculate TST by analyzing the recorded EEG signal. However, the SR20C does not record the EEG signal; the TST cannot be directly analyzed by the algorithm. Thus, the TST was replaced by the automatic modified time in bed (TIB) or was manually adjusted by the physician. The fourth factor is that an arousal event needs to be analyzed by the EEG signal. The Alice5 PSG can record the EEG signal, but the SR20C cannot.

The diagnosis results regarding SAHS from this algorithm for the 28 sleep data indicate that one case is normal ($AHI \leq 5$), seven cases are slight ($5 < AHI \leq 15$), eight cases are intermediate ($15 < AHI \leq 30$), and twelve cases are severe ($AHI > 30$). The sensitivity and PPV of auto-diagnosis are 92.3% and 92.3%, respectively.

4.3. Results compared to manual visual inspection

The Alice5 PSG analysis result itself cannot be absolutely correct. Therefore, to verify the effectiveness and accuracy of the algorithm, each event automatically analyzed by this algorithm was manually and visually inspected according to the 2012 AASM criteria.

All 30 auto analysis results are based on the 2012 AASM criteria and a 2s duration of the FSTS. A total of 8470 sleep respiratory events were

Table 4
Auto analysis results compared to the manual inspection.

		Predicted Condition			
		PA	NA	PH	NH
True Condition	Positive	5056	154	3088	67
	Negative	315	0	56	0
Total	Sensitivity	97.6%			
	PPV	95.7%			

identified in these datasets by computer auto analysis. There are 5328 apnea and 3142 hypopnea predicted events in all respiratory events.

In Table 4, PA is positive apnea, NA is negative apnea, PH is positive hypopnea, and NH is negative hypopnea. NA and NH in a true positive condition include misjudged or missing respiratory events by auto analysis according to the human inspection. Because the computer and humans both did not identify non-respiratory events, NA and NH for the true negative conditions are zero.

On the basis of the manual inspection, the total sensitivity and PPV of determining the respiratory event in the algorithm are 97.6% and 95.7%, respectively.

5. Discussion

According to the 2012 AASM criteria [34], the respiratory signal, oximetry signal and electroencephalograph (EEG) signal all together are needed for determining the apnea and hypopnea events. Except for the EEG signal, the other two signals are more easily obtained in the application. There are many different simple methods that only rely on one signal among the oximetry, respiratory and electrocardiograph (ECG) signals for determining AHI and diagnosing the SAHS.

The first class of methods depends on a single oximetry signal. From the point of view of signal acquisition and analysis, these methods are the most simple. One study pointed out that the correlation coefficient of the PSG-derived AHI and oximeter-derived respiratory disturbance index (RDI) was 0.97 and that the sensitivity and specificity were 98% and 88%, respectively [21]. Another study depicted the computer program-detected apnea event with a sensitivity of 97.9%; the predictive value of a computer-detected event was 90.8% [22]. However, another paper noted the poor diagnostic accuracy by using screening oximetry. The method was most successful only in detecting SAHS with a high likelihood of having OSA or those with more severe disease [23]. Relative to this paper's method, the classes of methods that only rely on the oximetry signal have obtained an unsteady, inaccurate result for diagnosing SAHS.

The second class of methods relies on a single respiratory signal. One study that used a support vector machine (SVM) classifier obtained a good result. In the method, the accuracy of determining the respiratory events reached 96% [24]. Another study used artificial neural networks for recognition of the respiration pattern. In the study, the average detection performance was over 90% [26]. Another paper used nasal pressure signal for real-time detecting sleep apnea and hypopnea events regardless of AHI severity with a sensitivity of 86.4%, and a positive predictive value of 84.5% [28]. In contrast to this paper, the above methods based on one respiratory signal have a similar accuracy but are more complex.

The third class of methods relies on a single ECG signal. Relatively, the ECG signal acquisition is very simple and mature. One study used the mean absolute deviations of the RR intervals and the SVM classifier to classify the OSA. The total accuracy of classifying the OSA was 77% in the method [29]. Another study that used the SVM classifier also obtained an average accuracy of 97.41%, a mean sensitivity of 99.16% and a mean specificity of 90.91% for OSA diagnosis [30]. Recent paper that used LS-SVM classifier with the Gaussian radial basis function (RBF) kernel to classify events of apnea and hypopnea together according to single-lead ECG signal, it achieved an accuracy of about 84% [31]. Another paper that used normal inverse Gaussian (NIG) modeling in the tunable-Q

factor wavelet transform (TQWT) domain for computer-assisted sleep apnea diagnosis obtained a sensitivity of 82% and a specificity of 91% [32]. In general, using an ECG signal to classify the OSA was an indirect and exploratory analysis method.

It is worthwhile to note that all of the above methods and this paper's method used different original sleep data. Therefore, direct comparison of different results only has reference significance. However, the methods based on oximetry or an ECG signal cannot classify the apnea type, such as obstructive apnea (OA), central apnea (CA) and mixed apnea (MA), because of the lack of a respiratory signal.

6. Conclusions

In this algorithm, the influence of systemic and sporadic changes in the respiratory amplitude was eliminated by using a sliding window and a FSTS. Therefore, the accuracy of judging respiratory events is improved and simultaneously improves the accuracy of obtaining the AHI.

The algorithm not only accurately identifies the respiratory events but also obtains other indicators to aid in the diagnosis of SAHS, such as the maximum and average duration of the respiratory events. If the nasal airflow and the thoracic and abdominal respiratory motions are simultaneously analyzed using this algorithm, the apnea events may be further classified into more valid OA, CA and MA events. These factors may help physicians obtain additional diagnostic information to correctly diagnose SAHS.

The algorithm is a simple, accurate and effective method for diagnosing SAHS. It is suitable for simple portable sleep screeners.

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References

- [1] Terry Young, M. Palta, J. Dempsey, J. Skatrud, S. Weber, S. Badr, The occurrence of sleep-disordered breathing among middle-aged adults, *N. Engl. J. Med.* 328 (1993) 1230–1235.
- [2] Paul E. Peppard, Terry Young, Jodi H. Barnett, Mari Palta, Erika W. Hagen, Khin Mae Hla, Increased prevalence of sleep-disordered breathing in adults, *Am. J. Epidemiol.* 177 (9) (2013) 1006–1014.
- [3] Terry Young, Paul E. Peppard, Daniel J. Gottlieb, Epidemiology of obstructive sleep apnea a population health perspective, *Am. J. Respir. Crit. Care Med.* 165 (2002) 1217–1239.
- [4] F.J. Nieto, T.B. Young, B.K. Lind, E. Shahar, J.M. Samet, Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study, *JAMA* 283(14) (2000) 1829–1836.
- [5] E. Shahar, C.W. Whitney, S. Redline, E.T. Lee, Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the sleep heart health study, *Am. J. Respir. Crit. Care Med.* 163 (2001) 19–25.
- [6] M.S. Ip, B. Lam, M.M. Ng, W.K. Lam, Obstructive sleep apnea is independently associated with insulin resistance, *Am. J. Respir. Crit. Care Med.* 165 (2002) 670–676.
- [7] Punjabi NM1, J.D. Sorkin, L.I. Katzel, A.P. Goldberg, Sleep-disordered breathing and insulin resistance in middle-aged and overweight men, *Am. J. Respir. Crit. Care Med.* 165 (2002) 677–682.
- [8] Jason M. Golbin, Virend K. Somers, Sean M. Caples, Obstructive sleep apnea, cardiovascular disease, and pulmonary hypertension, *Proc. Am. Thorac. Soc.* 5 (2008) 200–206.
- [9] Thunyarat Anothaisintawee, Sirimon Reutrakul, Eve Van Cauter, Ammarin Thakkinstian, Sleep disturbances compared to traditional risk factors for diabetes development: systematic review and meta-analysis, *Sleep. Med. Rev.* 20 (2016) 11–24.
- [10] John S. Floras, Sleep apnea and cardiovascular risk, *J. Cardiol.* 63 (2014) 3–8.
- [11] C. Gonzaga, A. Bertolami, M. Bertolami, C. Amodeo, D. Calhoun, Obstructive sleep apnea, hypertension and cardiovascular diseases, *J. Hum. Hypertens.* 29 (2015) 1–8.
- [12] David Lam, Karen SL. Lam, S.M. Mary, Obstructive sleep apnoea, insulin resistance and adipocytokines, *Clin. Endocrinol.* 82 (2014) 1–8.

- [13] H. Klar Yaggi, John Concato, Walter N. Kernan, Judith H. Lichtman, Obstructive sleep apnea as a risk factor for stroke and death, *N. Engl. J. Med.* 353 (2005) 2034–2041.
- [14] F. Javier Nieto, Paul E. Peppard, Terry Young, Laurel Finn, Sleep-disordered breathing and cancer mortality, *Am. J. Respir. Crit. Care Med.* 186 (2) (2012) 190–194.
- [15] F. Barbé, J. Pericas, A. Munoz, L. Findley, J.M. Anto, A.G.N. Agusti, Automobile accidents in patients with sleep apnea syndrome, *Am. J. Respir. Crit. Care Med.* 158 (1998) 18–22.
- [16] C.F.P. George, Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal CPAP, *Thorax* 56 (2001) 508–512.
- [17] J.A. Horne, L.A. Reyner, Sleep related vehicle accidents, *Br. Med. J.* 310 (1995) 565–567.
- [18] A. Arita, R. Sasanabe, R. Hasegawa, et al., Risk factors for automobile accidents caused by falling asleep while driving in obstructive sleep apnea syndrome, *Sleep* 19 (4) (2015) 1229–1234.
- [19] Daniel E. Jonas, Halle R. Amick, Cynthia Feltner, Rachel Palmieri Weber, Screening for obstructive sleep apnea in adults evidence report and systematic review for the US Preventive Services Task Force, *JAMA* 317 (4) (2017) 415–433.
- [20] R. Nisha Aurora, Nirupama Putcha, Rachel Swartz, Naresh M. Punjabi, Agreement between results of home sleep testing for obstructive sleep apnea with and without a sleep specialist, *Am. J. Med.* 129 (7) (2016) 725–730.
- [21] Juan-Carlos Vázquez, Willis H. Tsai, W. Ward Flemons, Akira Masuda, Automated analysis of digital oximetry in the diagnosis of obstructive sleep apnoea, *Thorax* 55 (2000) 302–307.
- [22] C.F. George, T.W. Millar, M.H. Kryger, Identification and quantification of apneas by computer-based analysis of oxygen saturation, *Am. Rev. Respir. Dis.* 137 (5) (1988) 1238–1240, 2000.
- [23] Lawrence J. Epstein, Gina R. Dordic, Cost-effectiveness analysis of nocturnal oximetry as a method of screening for sleep apnea-hypopnea syndrome, *Chest* 113 (1) (1998) 97–103.
- [24] Bijoy Laxmi Koley, Debangshu Dey, Automatic detection of sleep apnea and hypopnea events from single channel measurement of respiration signal employing ensemble binary SVM classifiers, *Measurement* 46 (2013) 2082–2092.
- [25] H. Nakano, T. Tanigawa, T. Furukawa, S. Nishima, Automatic detection of sleep-disordered breathing from a single-channel airflow record, *Eur. Respir. J.* 29 (2007) 728–736.
- [26] Péter Várady, Tamás Micsik, Sándor Benedek, Zoltán Benyó, A novel method for the detection of apnea and hypopnea events in respiration signals, *IEEE T. Bio-Med. Eng.* 49 (9) (2002) 936–942.
- [27] Nir Ben-Israel, Ariel Tarasiuk, Yaniv Zigel, Obstructive apnea hypopnea index estimation by analysis of nocturnal snoring signals in adults, *Sleep* 35 (9) (2012) 1299–1305.
- [28] Hyoki Lee, Jonguk Park, Hojoong Ki, Kyoung-Joung Lee, New rule-based algorithm for real-time detecting sleep apnea and hypopnea events using a nasal pressure signal, *J. Med. Syst.* (2016), <http://dx.doi.org/10.1007/s10916-016-0637-8>.
- [29] Bülent Yölmaz, Musa H. Asyaló, Eren Arökan, Sinan Yetkin, Fuat Özgen, Sleep stage and obstructive apnea epoch classification using single-lead ECG, *BioMed Eng. OnLine* (2010), <http://dx.doi.org/10.1186/1475-925X-9-39>.
- [30] Lili Chen, Xi Zhang, Changyue Song, An automatic screening approach for obstructive sleep apnea diagnosis based on single-lead electrocardiogram, *IEEE T. Autom. Sci. Eng.* 12 (1) (2015) 106–115.
- [31] Hemant Sharma, K.K. Sharma, An algorithm for sleep apnea detection from single-lead ECG using Hermite basis functions, *Comput. Biol. Med.* 77 (2016) 116–124.
- [32] Ahnaf Rashik Hassan, Computer-aided obstructive sleep apnea detection using normal inverse Gaussian parameters and adaptive boosting, *Biomed. Signal Proces.* 29 (2016) 22–30.
- [33] Diego Alvarez-Estevéz, Vicente Moret-Bonillo, Computer-assisted diagnosis of the sleep apnea-hypopnea syndrome: a review, *Sleep. Disord.* 2015 (2015) 33. Article ID 237878.
- [34] Richard B. Berry, Rohit Budhiraja, Daniel J. Gottlieb, Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events, *J. Clin. Sleep. Med.* 8 (5) (2012) 597–619.
- [35] Zhang Yanjun, Zhang Xiangmin, Yan Chengwen, Liu Wenhui, Correlation study in respiration fluctuations during sleep stages, *Technol. Health Care* 22 (6) (2014) 885–894.
- [36] Sven Rostig, Jan W. Kantelhardt, Thomas Penzel, Werner Cassel, Nonrandom variability of respiration during sleep in healthy humans, *Sleep* 28 (4) (2005) 411–417.

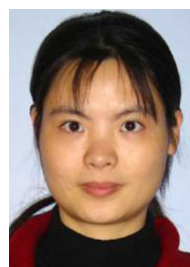
- [37] Alice S.L. Ng, Thomas K.S. Wong, M.D.I. Gohel, Winnie W.M. Yu, Using pulse oximetry level to indicate the occurrence of sleep apnoea events, *Stud. Health Technol. Inf.* 122 (2006) 672–675.



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