

# Screening for Obstructive Sleep Apnoea Based on the Electrocardiogram - The Computers in Cardiology Challenge

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## Abstract

*We present a method of screening for obstructive sleep apnoea based on the electrocardiogram (ECG). The algorithm combines information from the ECG-derived respiration (EDR) signal and the RR interval tachogram. Power spectral features from the EDR signal were computed using the discrete harmonic wavelet transform, considering the power at the respiratory frequency and at frequencies below 0.1Hz. Cycles of tachy/bradycardia (consistent with an arousal from sleep, as would be expected at the end of an episode of apnoea) were identified from the RR interval tachogram. Features were collated into minute-by-minute vectors and passed to a classifier. The algorithm correctly classified 81% of all minutes in the test database, with 29/30 patients correctly identified as apnoea or normal. Visual classification produced 92% correct classification, with all 30 patients correct.*

## 1. Introduction

The obstructive sleep apnoea/hypopnoea syndrome (SAHS) has been estimated to affect up to 4% of middle-aged males and 2% of middle-aged females [1]. The syndrome is characterised by repetitive episodes of airway narrowing or collapse during sleep, with each episode terminating in arousal and resumption of normal breathing. The resulting sleep fragmentation causes daytime sleepiness, with consequences such as increased risk of road traffic accidents [2]. The SAHS also carries an increased risk of hypertension, although the mechanisms underlying the association between the two are not yet clear [3, 4]. Polysomnography is traditionally used to diagnose the SAHS; however, this is both time-consuming and expensive, and a cheaper diagnostic test (commonly overnight oximetry) is frequently used to reduce the load on a sleep laboratory. In this paper, we show how information derived from the electrocardiogram (ECG) can be used to screen for the SAHS.

The arousal which terminates each episode of apnoea

or hypopnoea elicits the orienting reflex [5], causing an increase in heart rate and blood pressure. Measurements of such autonomic responses can provide a sensitive marker of arousals from sleep [6], and heart rate measurements can easily be made from the ECG. However, autonomic arousals are not exclusive to the SAHS, as similar patterns are seen in other sleep disorders (such as periodic limb movement syndrome [7]), and in response to spontaneous arousal [6].

The ECG offers additional information which may be used in screening for the SAHS. Respiration causes changes in the ECG electrode position on the chest (with respect to the heart) and also changes in thoracic impedance. These changes cause a well-known change in the electrical axis of the ECG which is closely correlated with respiration [8]. This signal has previously been applied to the detection of the SAHS [9, 10], and is denoted here as the ECG-derived respiration (EDR) signal.

The algorithm outlined in the next section combines information from both the EDR signal and autonomic arousals.

## 2. Methods

### 2.1. Subject data

The algorithm was evaluated on data from the Computers in Cardiology 2000 Challenge, available from the Physionet website [11]. This data set consists of overnight, single channel ECG recordings (sampled at 100Hz) obtained from 70 patients. Thirty-five recordings are designated as training data, and minute-by-minute annotations (denoting apnoeic or normal breathing during each minute) are available for these recordings. The remainder of the recordings make up the test data set. Both the training and test sets contain recordings from 20 patients with moderate to severe obstructive sleep apnoea, 10 control patients, and 5 "borderline" patients (for further details of the data and for category definitions see the Physionet website).

## 2.2. Signal preprocessing

The time of each heartbeat was identified from the ECG using a QRS template-matching algorithm followed by automatic and manual correction.

The EDR signal is conventionally derived from measurements of the QRS complex [9], which corresponds to the rapid contraction of the ventricles as blood is expelled from the heart. Due to the low sampling rate of these data combined with the relatively fast changes in the ECG during the QRS complex, this feature was found to give a poor estimation of respiratory activity. We instead opted to base the EDR on measurements of the T-wave, which corresponds to the slow repolarisation of the ventricles after contraction. T-waves were assumed to fall within a fixed window (typically 15-40ms) following each QRS complex. Each signal segment within this window was linearly detrended and the average absolute value of the segment retained as the value of the EDR signal at that time.

The RR interval tachogram was constructed by taking the difference between consecutive beat times. This signal was low-pass filtered to remove respiratory sinus arrhythmia.

## 2.3. Feature extraction

The discrete harmonic wavelet transform (DHWT) [12] was used to characterise the time-frequency properties of the EDR signal. The DHWT has the desirable property that each wavelet scale (termed a "level") represents a unique frequency band. In order to compute the DHWT, the Fourier coefficients of the EDR signal are first required. However, the EDR signal is unevenly sampled (samples are available only at the time of each T-wave). The non-equispaced Fourier transform [13] was therefore used to compute the Fourier coefficients. A sampling rate of 1.2Hz was used as this yields DHWT levels which are convenient for respiratory analysis. The DHWT has been successfully applied to the identification of moderate to severe SAHS based on the RR interval signal [14].

The DHWT time-frequency map was normalised with respect to total power at each time slice. Since we are interested in the relative powers in various bands of the frequency spectrum, the absolute magnitude of the EDR signal is unimportant.

Each episode of apnoea or hypopnoea causes a swing in the baseline of the EDR signal (see Figure 1); one of the signal components of interest is therefore at the rate of episode repetition, generally between 15-60s (0.017-0.07Hz). This corresponds to levels 11 and 12 of the DHWT of the EDR signal, which cover the frequency band 0.01875-0.075Hz. The termination of a respiratory event is also often accompanied by compensatory hyperventilation, which causes a burst of power in the DHWT at the respiratory frequency. Level 15 of the EDR DHWT

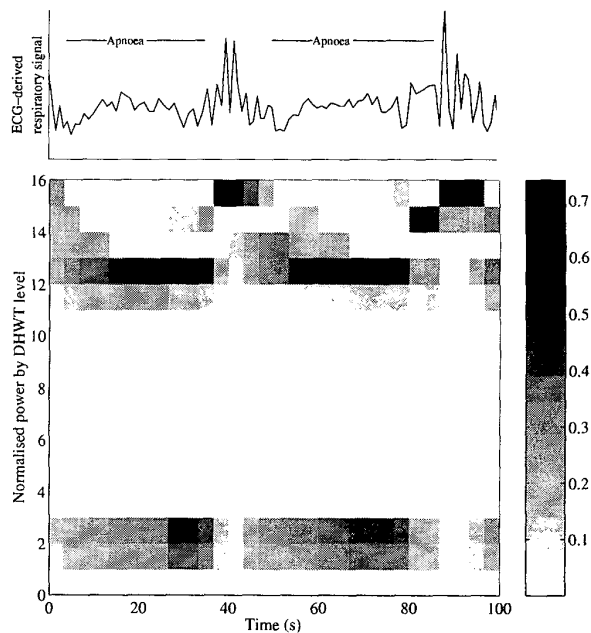


Figure 1. An example of the ECG-derived respiratory signal during apnoea and its discrete harmonic wavelet transform.

(which covers the frequency band 0.3-0.6Hz) was therefore processed using a second DHWT in order to identify bursts recurring at 15-60s intervals.

The orienting reflex causes an increase in heart rate and blood pressure at the termination of each respiratory event. In turn, the rise in blood pressure elicits a decrease in heart rate, so that arousals are marked by a tachy/bradycardia cycle in heart rate. Such arousals were identified on the basis of the slope of the RR interval signal. A decrease of 6ms per second, sustained over a 10s window, was considered to be a significant tachycardia, provided it was followed within 20s by a bradycardia of similar dimensions. The time of occurrence and amplitude of each tachy/bradycardia cycle were noted.

Features were assembled into vectors, with one four-element vector per minute of each recording. For minute  $k$  of a recording, the first two feature vector elements were the median powers in levels 11 and 12 of the EDR DHWT during that minute. If the sum of these two powers exceeded 20% of the median signal power during minute  $k$ , then evidence of an arousal was sought during minute  $k$  and the first half of minute  $k + 1$ . The maximum power related to compensatory hyperventilation and the amplitude of the largest tachy/bradycardia during that time were taken as arousal features. In the event that no tachy/bradycardia cycle occurred during a given minute, a value of zero was used for the last element.

The feature vectors were passed to a mixture model classifier [15]. Once each minute in a given test recording had been classified, an overall label of “apnoea” or “normal” was assigned to that recording. Test recordings with less than 40 total minutes of apnoea (approximately 5 apnoeas per hour) were considered to be normal.

### 3. Results and discussion

For the minute-by-minute classification of the test set recordings, an accuracy of 81% was obtained (14052/17268 correct classifications). The labelling of each test patient gave 29/30 correct overall classifications, with one control subject incorrectly labelled as apnoea.

Subsequent manual editing of the minute-by-minute classifications improved the results significantly, to 92% accuracy (with all 30 patients correctly labelled).

The majority of the classification errors produced by the algorithm probably stemmed from episodes of hypopnoea. Whereas an apnoea is a near-complete cessation of airflow, an obstructive hypopnoea is a partial reduction in airflow amplitude caused by airway narrowing (but not total collapse). Since respiration remains partially intact, the EDR signal during an episode of hypopnoea can be expected to show a component at the respiratory frequency (0.3-0.6Hz). Figure 2 shows an example of the type of data which was frequently misclassified as being normal by our algorithm. Since the EDR signal shows a strong component at the respiratory frequency (approximately 0.4Hz), this data segment was probably recorded during a period of recurring hypopnoeas. The training set annotations made no distinction between apnoea and hypopnoea, so this could not be confirmed.

There were further difficulties with the classification performance of the algorithm which were probably caused by hypopnoeas. The definition used for hypopnoea in the competition data was as follows: intermittent drops in respiratory flow below 50%, accompanied by drops in oxygen saturation of at least 4%, and followed by compensating hyperventilation. It is known that 4% dips in saturation are an insensitive marker of the SAHS [16], and recent guidelines [17] recommend a less stringent definition of hypopnoea.

The EDR signal and autonomic arousals shown in Figure 3 suggest two separate, short periods of disordered breathing (and indeed our algorithm classified both periods as apnoea). However, the annotations for this recording deny disordered breathing during the second period. It is possible that the respiration patterns during the second period did not quite meet the definition of hypopnoea and were therefore scored as normal. Our algorithm may be more sensitive to the SAHS than the polysomnography-derived annotations.

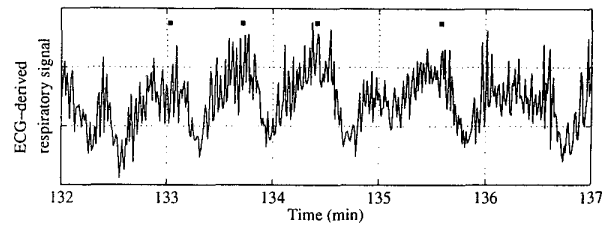


Figure 2. An example of data which was frequently misclassified as normal by the algorithm. The ECG-derived respiratory signal shows large baseline swings but strong activity at the respiratory frequency (black squares indicate times of autonomic arousals).

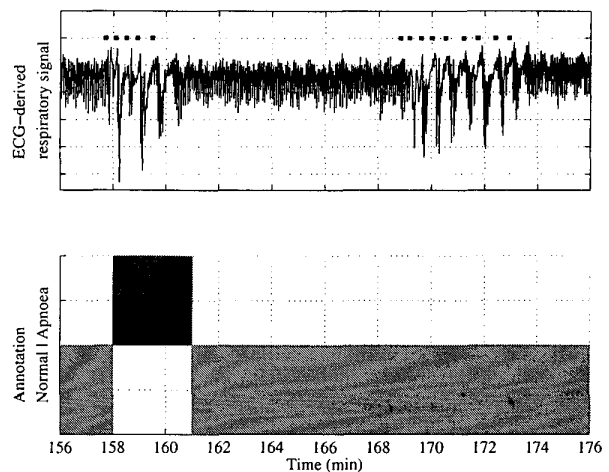


Figure 3. An example of ambiguity in the diagnostic signals. The ECG-derived respiratory signal (top; black squares indicate times of autonomic arousals) suggest respiratory events from time 158-161 and from 170-174. However, the annotations (derived from the polysomnogram) confirm respiratory events only from 158-161.

### 4. Conclusions

The performance of this and other algorithms developed for the Computers in Cardiology 2000 Challenge is an encouraging step toward reliable screening for the SAHS using the ECG. However, the nature of the test subjects should be kept in mind: most displayed moderate to severe SAHS which is generally well-characterised by oximetry alone. Oximetry may well have given similarly good results and with lighter computational requirements (oximetry data for the test subjects were not available at the time of writing).

The sensitivity of oximetry in screening for the SAHS is known to be poor [16]; the performance of the current

approach when applied to mild SAHS patients is yet to be seen. However, there is good reason to be optimistic, since the arousal response which accompanies the termination of each episode of disordered breathing is similar regardless of syndrome severity. The diagnostic value of the EDR signal in mild SAHS is not known. Replacing the single ECG lead with two orthogonal leads may be necessary, as this is known to improve the accuracy of the derived EDR signal [9].

Screening using the ECG rather than oximetry has one clearly favourable point: the fact that the ECG has been recorded would allow a cardiological screening test to be carried out using the same data. Cardiovascular comorbidities are common in the SAHS [18], and a pre-polysomnography ECG is commonly carried out as a routine component of the diagnostic protocol for the SAHS [19].

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