Polysomnographic Sleep Recording with Simultaneously Acquired 12 Lead ECGs: A Study for Detection and Validation of Apnea Related ECG Changes

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Abstract
The aim of our study was to investigate whether morphological ECG parameters are changed during sleep disorders, in particular during apnea. We have set up jointly with the sleep laboratory of Marburg University a new database with polysomnographic and simultaneously recorded 12 lead ECG data. Analyses results on single cases and on a data set of 9 patients are presented. They confirm our hypothesis that morphologic ECG parameters vary systematically with apnea phases. While mean values differences seem to be small between regular and apnea respiration, standard deviations of these parameters are marked. From a separate analysis of data for each type of apnea and a joint processing of heart rate variability we expect additional insight into the reasons for the morphologic ECG changes.

1. Introduction
Wittchen reported about a recent study NISAS 2000 (Nationwide Insomnia Screening and Awareness Study), performed in cooperation between the Max-Planck-Institute for psychiatry in Munich and Technical University Dresden. 20,000 patients from 539 German general practitioners have been examined 42% of the patients suffered from sleep disorders but only 70% of these cases have been detected by the GPs. 21% of the patients did have difficulties to fall asleep, 27% did wake up during night several times. Obstructive sleep apnea has been found in 17% of the patients. 26.7% of all general practitioner patients fulfilled the internationally defined criteria for insomnia. Risks associated with sleep apnea are, e. g. hypertension and sleep attacks during the day. It is not surprising that therefore diagnoses and therapy of sleep apnea gain increasing interest [1].

Early detection of sleep apnea may be possible by using ambulatory recording methods, e.g., long-term ECG recording, long-term blood pressure, and dedicated recording systems for acquisition for respiratory flow, (leg, body) movement, blood oxygen saturation, heart rate and snoring. The final assessment of sleep apnea must be done in a sleep laboratory using cardio-respiratory polysomnography [2,3]. Polysomnographia includes the recording of the EEG, EOG and EMG to determine sleep stages. Furthermore the oronasal air flow, chest wall and abdominal wall movements for respiratory effort, oxygen saturation to monitor the effect of respiration and at least one ECG channel for heart rate analysis and arrhythmia detection.

For the Computers in Cardiology conference 2000, a challenge was made to recognise and quantify sleep apnea from single channel ECGs provided for altogether 70 recordings [4].

The results were surprisingly good (sensitivity and specificity reach in the best analysis more than 95%).

A meta-analysis of the performance results revealed that the best results were obtained by methods where also morphological parameters of the single ECG have been included in the analysis[5].

We have therefore set up a joint study with the sleep laboratory of the Philipps University Marburg/Germany to systematically investigate ECG parameters and their changes associated with sleep disorders, in particular, obstructive sleep apnea.

2. Material and methods
2.1. Setting up the data base
To study the phenomena a new data base had to be set up. The data base consists of extensive polysomnographic recordings, including EEG, EOG, EMG, nasal airflow, measurement of the respiratory efforts from abdomen and ribcage, oxygen saturation, snoring noise and in case of therapy also recording of the nasal continuous positive airway pressure (CPAP). In parallel to these polysomnographic recordings the conventional ECG with 8 independent leads were acquired by the HES LKG system. This system consists of a stand alone ECG amplifier and a PC and is described in this issue of the Computers in Cardiology proceedings elsewhere.

So far the polysomnographic data and the ECG data are acquired on two different computers. To synchronise the recordings the time stamps of the system time within the computers are used for identification of data segments. Each sleep recording results in 400-600 MBytes of data.
2.2. Examples of data analysis

For analysis and display of the polysomnographic recordings the program WINDAY developed at Marburg University has been used. For analysis and display of the ECG data, the program system HES LKG developed at Medical School Hannover has been used.

Figure 1 Example for a polysomnographic data display

Figure 1 shows a sample of signals for a reference interval of 5 min. at the beginning of a polysomnographic record during the bed time before the patient falls asleep. The most upper channel is the ECG, the next channel below the nasal airflow, the third channel is the thorax movement (ribcage), then the movement of the abdomen is shown, below the oxygen saturation and on the bottom trace the heart rate. The signal in all channels shows a smooth time course with a very regular rhythm.

Figure 2 Example of ECG data from the same Reference phase.

The tracings of the heart rate, of the R-amplitude, in lead I and the R/S amplitude ratio in lead V4 show an almost constant level and a very smooth time course.

Figure 3 and 4 show data of the same patient for a period of obstructive apnea.

Figure 3 5 min. Polysomnographic record sample of an obstructive apnea phase with annotation

Figure 4 Behaviour of ECG parameters during the apnea phase shown in fig. 3.

The same channels as in figure 1 are shown. On the second channel the oscillations and cessations of the nasal airflow can be seen. These seven phases are annotated as “obstructive apnea” OA. While in the thorax signal during the cessations small regular oscillations can be still observed the ribcage signal of the abdomen is almost flat with low amplitude oscillations opposite in amplitude direction to the thorax signal. The tracing on the oxygen saturation shows the lowest value just after the apnea. This phase shift is due to time lag between the drop in the oxygen saturation measured by a finger pulse oximeter. The numbers in the boxes below give the degrees oxygen desaturation ranging from 5-10%. The
trace on the bottom shows the almost periodic decrease and increase of the heart rate following the breathing behaviour.

The traces on figure 4 show again the heart rate, the R-amplitude in lead I and the R/S-amplitude ratio (x100) in lead V4 for the apnea interval as above. Clearly the modulation of the morphologic ECG parameters by the obstructive apneas can be seen. It should be noted that the R-amplitude in I and the amplitude ratio have the opposite phase to the heart rate changes.

Another illustration on the apnea related morphologic ECG changes is given on the following figures. The data refer to a 26 years old male patient with 122 obstructive apneas with an average duration of 25.5s during sleep between 22:15 hr – 02:37 hr during sleep.

At 02:45am he got treatment with Continuous Positive Air Pressure (CPAP), mean pressure 7.6cm H\textsubscript{2}O. Within the following sleeping phase of approx. 3 hrs the patient have only one OA of 13s.

<table>
<thead>
<tr>
<th>Time</th>
<th>Duration in min.</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>23:59-0:30</td>
<td>31</td>
<td>Obstr. Hypopnea</td>
</tr>
<tr>
<td>0:31-0:34</td>
<td>3</td>
<td>Obstr. Apnea</td>
</tr>
<tr>
<td>0:37-1:16</td>
<td>39</td>
<td>Obstr. Apnea</td>
</tr>
<tr>
<td>1:29-1:35</td>
<td>6</td>
<td>Obstr. Snoring</td>
</tr>
<tr>
<td>1:35-1:39</td>
<td>4</td>
<td>Obstr. Hypopnea</td>
</tr>
<tr>
<td>2:45-6:20</td>
<td>215</td>
<td>regul. Respiration</td>
</tr>
</tbody>
</table>

Figure 5 Apnea phase patient R.T., polysomnographia.

Full cessations of the nasal airflow can be seen. Oxygen desaturation reaches 7%

Figure 6 Apnea associated ECG parameter changes

Very distinct HR and R/S changes in V3 can be seen!

Table 1: Respiratory Annotation Patient R.T.

Figure 7 60 min. polysomnographic record of Patient R.T. during CPAP.

A smooth and regular periodic thorax and abdomen movement and a constant 100% oxygen saturation can be seen

Figure 8 ECG record of Patient R.T. during CPAP

Heart rate as well as the ECG parameters reflect the “stable” function of the cardio-respiratory system during treatment.
3. Analysis results on a data set

Up to now 50 night-recordings have been performed from 40 patients, 38 males, 2 females, main age 54.4 years. Out of this nine patients, 9 males, main age 52.7 with high signal quality have been selected for a more comprehensive analysis of ECG parameters. Altogether data from 2:42 hrs of regular respiration which results in 972 measurements/parameter and for 9:47 hrs of apnea resulting in 3.522 measurements per parameter have been evaluated.

The two following diagrams show mean values and standard deviations of heart rate, the R-Amplitude in V5, STT-Integral in V5, the R/S Ratio in V3, the R-Amplitude in V4, the S-Amplitude in V2, the QRS frontal vector magnitude, the QRS frontal angle and the T frontal vector magnitude.

![Mean of ECG Parameters](image1)

Figure 9 Mean Values of ECG parameters during regular respiration and at apnea.

While the mean values of R-waves in V5, V4 and S-wave in V2 in apnea phases increase the vector magnitudes seem to decrease.

![Standard Deviations of ECG Parameters](image2)

Figure 10 Standard Deviations of ECG parameters during regular respiration and at apnea.

More clearly the standard deviations of the respective ECG parameters are enlarged in all parameters during the apnea phases. This confirms our observation from figures 2-8 that also morphologic ECG parameters are “modulated” during the apnea phases.

4. Discussion and conclusion

Our pilot analysis has revealed that indeed morphological ECG parameters are affected by apnea phases. ECG parameters may behave differently among patients and leads, e.g. sometimes the frontal vector magnitude shows a clear modulation with the apnea respiration but in other cases this could not be observed although other parameters change.

It is at this stage of our understanding possible that the morphologic ECG changes may be caused by both, positional changes of the heart during apnea-affected breathing as well as depolarisation and repolarisation alterations associated with the oxygen saturation changes during the apnea. Systematic effects on ST-T have not been found so far.

The global annotation of apnea (hyponea, mixed apnea, central apnea and obstructive apnea) is too coarse. In the next step the ECG will be separately analysed for the different types of apneas.

In a next step jointly heart rate variability and morphologic ECG parameters need to be analysed, e.g. for correlation, coherence, frequency spectrum etc.

References


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