

PERIPHERAL ARTERIAL TONOMETRY MONITORS CHANGES OF AUTONOMOUS NERVOUS SYSTEM IN SLEEP APNEA

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Abstract – The diagnosis of obstructive sleep apnea requires assessment of cardiovascular function because sleep apnea has been recognized as a risk factor for cardiovascular disorders. We tested a new sensor which monitors autonomous nervous function by the measurement of peripheral arterial tone (PAT) on the finger. Twentyone patients with obstructive sleep apnea and arterial hypertension underwent polysomnography with parallel invasive blood pressure and PAT. The PAT signal allows to track hemodynamic changes and it can recognize subcortical arousals which accompany apneas. Correlation between PAT attenuations and cortical arousal was significant ($r=0.583$; $p<0.01$). The PAT signal cannot replace invasive blood pressure but provides additional information which allows to investigate changes of sympathetic and parasympathetic activities during sleep.

Keywords – Sleep apnea, peripheral arterial tone, autonomous nervous system, sympathetic tone

I. INTRODUCTION

A new sensor was recently introduced which reflects peripheral circulatory responses to disordered breathing [1] (fig. 1). This peripheral arterial tonometry (PAT) sensor selectively measures the arterial component of finger volume changes accompanying the pulse wave. These changes are an integral part of the hemodynamic response to obstructive sleep apnea related arousal. In order to directly relate these changes to hemodynamic changes we compared PAT and invasive blood pressure.

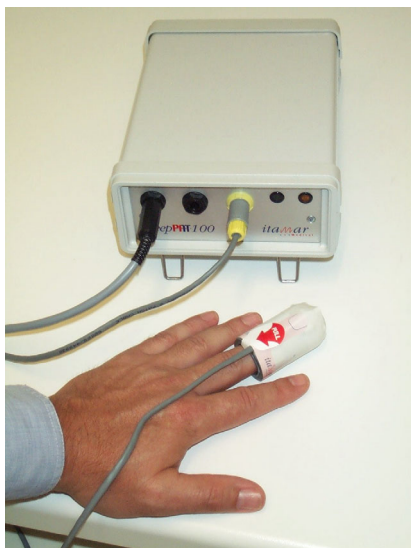


Fig 1. The PAT preamplifier and the PAT sensor attached to a finger. In order to start the recording, the red stripes on the sensor need to be pulled out. This inflates the internal cuff and prevents the sensor from falling off. The applied pressure is sub-diastolic and does not cause discomfort.

II. METHODOLOGY

Twenty-one patients with obstructive sleep apnea (AHI>20) and arterial hypertension according to WHO criteria ($> 140 / 90$ mmHg) were recruited for the comparative study. After an initial sleep study used to confirm the diagnosis of sleep apnea a second sleep study was performed with the additional recording of invasive arterial blood pressure and the PAT signal.

Sleep was recorded using an Embla polygraph (Flaga Inc.) together with a Hellige blood pressure amplifier and a Resptrace for inductive plethysmography (fig. 2). Sleep was evaluated according to Rechtschaffen and Kales [2], arousal were scored according to ASDA criteria [3].

Attenuations in the amplitude of PAT signal by at least 40% compared to baseline were counted visually and rescored by a second scorer in order to obtain inter-rater reliability. When scoring the PAT dips, both scorers were blinded to the respiratory signals and the blood pressure. Attenuations were classified as being isolated, or synchronized with a drop in oxygen saturation, or with an increase in heart rate or both. In addition double attenuation patterns were observed. Artifacts caused by major body movements were discarded from further analysis.

Computer based evaluation of blood pressure was done beat by beat and systolic, diastolic and mean values were derived. The amplitude of the PAT signal was calculated beat by beat. Heart rate was derived from the ECG.

III. RESULTS

Patients' age was 56.6 ± 9.2 years. Body mass index (BMI) was 33.7 ± 5.7 . The evaluation of polysomnography resulted in a total number of apneas and hypopneas of 319 ± 187 events per hour. If only the apneas were counted we obtained 210 ± 158 events per night. In addition an apnea-hypopnea index (AHI) was calculated as events per hour of total sleep time.

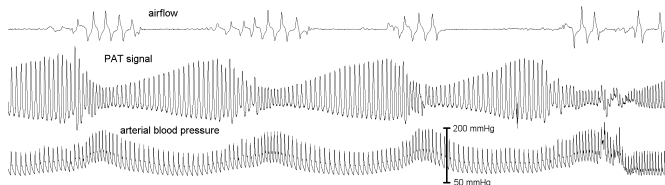


Fig 2. The recording example shows repetitive apneas with concomitant blood pressure swings and PAT amplitude swings. Apneas can be recognized by the cessation of airflow (top trace). The PAT amplitude attenuates during each single apnea (middle trace).

The total number of attenuations in the PAT signal (artefact movements removed) summed up to 275 ± 116 events. 221 ± 110 attenuations in the PAT signal were accompanied by oxygen desaturation and heart rate increases. Invasive blood pressure swings reflected both apnea and hypopnea events (fig. 2).

Small blood pressure swings at a short time scale in the range of respiratory rate also reflected snoring (pulsus paradoxus). The PAT signal did follow apnea related blood pressure changes in a complex way. The initial decrease in blood pressure at the beginning of an apnea was reflected as an increase in the PAT signal. The increase in blood pressure during an apnea often had no, or minor effects of the PAT signal. The final large increase of blood pressure during hyperventilation at the time of apnea terminating arousal was consistently well mirrored by a distinct drop in the PAT signal (fig. 2).

Correlation between all visually counted PAT attenuations of 21 subjects (only movement artifacts were removed from the number of PAT attenuations) and arousal (scored according to ASDA criteria) resulted in a Pearson correlation coefficient of $r=0.583$ ($p<0.01$). Correlation between PAT attenuations and apnea and hypopnea events was $r=0.656$ ($p<0.01$) even higher.

IV. DISCUSSION

Our study is the first comparison of the PAT signal with continuous invasive blood pressure recording during the night in patients with sleep related breathing disorders. The peripheral arterial tone measured by the PAT does reflect sympathetic activation. Sympathetic activation occurs at the end of each apnea and can be also seen in blood pressure and heart rate evaluations (fig. 3). Rapid-eye-movement sleep modulates the duration of apneas and also the effect of apneas on blood pressure, heart rate and PAT amplitude.

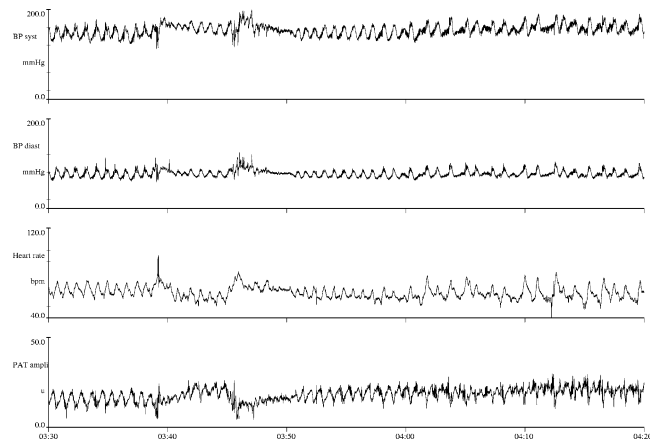


Fig 3. Fifty minutes of the traces of systolic blood pressure (1st curve top), diastolic blood pressure (2nd), heart rate (3rd) and computed PAT amplitude (4th) are plotted for a patient with severe sleep apnea. On the left side non-REM sleep is found and on the right side REM sleep is found. The regular cyclic behavior of PAT dips becomes much more irregular.

Our data suggest that there are still different mechanisms which regulate the three aspects investigated of sympathetic activation in the condition of sleep apnea. Aspects reflected in the PAT signal are peripheral blood pressure in the finger, vascular tone and stroke volume changes. The effects of stroke volume changes could be best observed through arrhythmias. These different effects again differs from cortical arousal found in EEG signals.

V. CONCLUSION

The PAT signal is a good marker for arousal with concomitant cardiovascular changes. The correlation between AHI and attenuations in the PAT signal did not confirm a uniform relation between apneas, hypopneas and cardiovascular arousals. The direct comparison with blood pressure indicates that the PAT signal reflects changes in sympathetic tone and stroke volume. Local vascular tone changes also influence the signal, so a direct relation between PAT and stroke volume cannot be expected. The fractions of these contributions remain unclear and may vary with time, also dependent on sleep stage. The PAT signal with the current conditioning cannot be used to predict changes in blood pressure in patients with sleep apnea.

Sleep related breathing disorders are common and have been recognized as a risk factor for arterial hypertension [4] and thereby for subsequent cardiovascular problems [5]. If sleep related breathing disorders are diagnosed at an early stage of disease effective therapy is possible and can prevent cardiovascular consequences. In order to achieve an early diagnosis of sleep related breathing disorders, we tested a new sensor, which measures peripheral arterial tone as an indicator for sympathetic activity, against the gold standard of sleep laboratory based polysomnography.

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