Ambulatory monitoring of blood pressure disturbs sleep and raises systolic pressure at night in patients suspected of suffering from sleep-disordered breathing

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1. The aim of the study was to assess the effect of ambulatory monitoring of blood pressure on sleep and on blood pressure in middle-aged patients.

2. Nine consecutive patients (seven men, two women; mean age 57 years) complaining of snoring and various degrees of excessive daytime somnolence were studied. Five patients were normotensive and four were being treated for hypertension. During one night standard laboratory polysomnography was performed with monitoring of blood pressure by a silent ambulatory monitor and continuous infrared blood pressure by photoplethysmography.

3. Ambulatory blood pressure significantly disturbs sleep architecture, causing EEG arousals in 64\% of measurements, and induces a significant rise in blood pressure during systolic pressure measurement by the ambulatory monitor (rise in systolic pressure, 13.7±15.9 mmHg, \(P<0.001\); rise in diastolic pressure, 3.7±8.2 mmHg, \(P<0.01\)). At the time of diastolic measurement, blood pressure had returned to the preinflation value. The rise in systolic blood pressure was higher when an arousal was associated with cuff inflation (\(P<0.001\)). This rise in blood pressure is probably the consequence of sympathetic nervous system activation.

4. We conclude that ambulatory blood pressure recordings of systolic blood pressure during sleep should be interpreted with caution as systolic blood pressure may be significantly increased in patients suspected of suffering from sleep-disordered breathing.

INTRODUCTION

Ambulatory blood pressure (ABP) monitoring is increasingly used to diagnose and to follow patients with high blood pressure [1–5]. A nocturnal fall in blood pressure is usually observed in normal and hypertensive patients [6–8], and the absence of this nocturnal decrease has been associated with cardiac hypertrophy [9, 10] and increased vascular risk [11–13]. The effect of ABP on sleep is controversial: Degaute et al. [14] found only a small decrease in sleep efficiency and in the amount of slow-wave sleep, whereas Schwan and Eriksson [15] reported awakenings in 67\% of measurements. These awakenings were related by the authors to the sound of the monitor and to the cuff pressure. However, not only awakenings but also transient arousals [16] have been associated with increases in blood pressure. These transient arousals have not been investigated in previous studies. Furthermore, several studies have concluded that ABP monitoring has no overall effect on recorded blood pressure [15–19]. However, these conclusions were not based upon simultaneous measurement of continuous blood pressure and sleep [17–19]. Recently, Davies et al. [20] reported appreciable arousals and alterations of blood pressure in six normal subjects during ABP monitoring, which they attributed to the noise of the devices, but no data have yet been reported on patients. Therefore, the aim of this study was to evaluate the effect of a silent ABP-monitoring device on transient arousals during sleep and on continuous blood pressure measured simultaneously in nine middle-aged patients.

METHODS

Subjects

This prospective study included nine consecutive patients, seven men and two women, complaining of snoring and various degrees of excessive daytime somnolence. All patients were referred to the sleep laboratory to be screened for sleep-disordered breathing. The mean age of the patients was 57 years (range 43–67 years) and mean body mass index (BMI) was 26.9 kg/m² (range 21.6–32.1 kg/m²). Five patients were normotensive [mean systolic blood pressure (SBP) measured supine before sleep, 118 mmHg (range 105–154 mmHg); mean diastolic blood pressure (DBP), 70 mmHg (range 60–85 mmHg)]. Four subjects were hypertensive, and treated by one (one subject), two (two subjects) or...
three (one subject) antihypertensive agents including a calcium antagonist, a diuretic, a β-adrenoceptor blocker and a converting enzyme inhibitor. The treatment was not interrupted for the study. Their mean SBP under treatment was 146 mmHg (range 120–185 mmHg) and their mean DBP was 91 mmHg (range 70–115 mmHg). All subjects gave their informed consent to the institutionally approved study.

Protocol

Each patient underwent polysomnography, which included a two-channel EEG, an ECG, an electro-oculogram (EOG) and chin and anterior tibialis electromyograms (EMGs) as well measurement of thoracic and abdominal movements (Respitrace), transcutaneous oxygen saturation (Ohmeda, Biox 3740), nasal flow by thermistor and non-invasive beat-to-beat finger arterial pressure by a Finapres 2300E device (Ohmeda, Boulder, CO, U.S.A.) placed upon the third finger of the left hand [21]. An ambulatory blood pressure monitor (Nippon Colin ABPM-630 Japan) was placed on the opposite arm. This device uses a carbon dioxide cartridge for arm cuff inflation and is therefore silent during the blood pressure measurement procedure [22]. Agreement within 5 mmHg between ABP and sphygmomanometric pressure was ascertained on three measurements on the same arm before beginning ambulatory measurement. The arm cuff pressure was also recorded on the polygraph using a Spectromed P23XL sensor and a Gould pressure processor (Gould, Ballainvilliers, France). The ABP monitor was applied to the patients between 18.00 and 19.00 hours and set to measure blood pressure every 30 min during the night. The oscillometric method was used for blood pressure determination by the Nippon Colin device. The Finapres device was automatically interrupted for 5 min every 30 min to avoid patient discomfort. These interruptions were scheduled to occur between ABP measurements. All variables were simultaneously recorded on a Schwarzer ED24 device (Madhaus, Germany) at a speed of 15 mm/s throughout the night. Positioning of the patients’ arms was continuously monitored by an infrared video camera to avoid any erroneous Finapres measurement resulting from a change in hydrostatic level.

Data measurements

Stage of sleep before (SS0) and during (SS1) ABP measurements was determined by visual analysis of the EEG, EOG and EMGs following a modified version of the criteria of Rechtschaffen and Kales [23] using 20 s epochs. Following the criteria of the American Sleep Disorders Association, EEG arousal was defined as an abrupt rise in EEG frequency (except spindles) for 3 s or more during non-rapid eye movement (REM) sleep and was also associated with increases in EMG activity during REM sleep [24]. The occurrence and duration of EEG arousals were noted. Similarly, stages of sleep and arousals were evaluated before and after finger cuff inflation at the end of the automatically interrupted period of the Finapres. Average SBP and DBP were measured on the Finapres recording, as shown in Fig. 1, for three 10 s intervals: before cuff inflation (P0); immediately after maximum cuff inflation (P1), corresponding to the time of ambulatory SBP determination; and immediately before the final rapid cuff deflation (P2) at the time of ambulatory DBP determination. This was performed manually by averaging continuous arterial pressure waves on the recordings. Heart rate was also averaged during these three intervals. This arbitrary duration of 10 s was chosen because it allows breathing variations in
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Table 1. Sleep quality and respiratory disturbances in nine patients. Total sleep time (TST), sleep efficiency, stages of sleep as a percentage of TST and apnoea-hypopnoea index (AHI) in the studied patients.

<table>
<thead>
<tr>
<th>Patient number</th>
<th>TST (min)</th>
<th>Sleep efficiency (%)</th>
<th>Stage 1 (%)</th>
<th>Stage 2 (%)</th>
<th>Stage 3 + 4 REM (%) of TST</th>
<th>AHI (per hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>268</td>
<td>0.72</td>
<td>10</td>
<td>62</td>
<td>1</td>
<td>26</td>
</tr>
<tr>
<td>2</td>
<td>364</td>
<td>0.83</td>
<td>3</td>
<td>58</td>
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<tr>
<td>3</td>
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<td>0.85</td>
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<td>19</td>
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<tr>
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<td>38</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
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<td>26</td>
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<tr>
<td>6</td>
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</table>

Blood pressure to be averaged over at least two respiratory cycles and is long enough to include the precise times of measurement of SBP and DBP by the ABP monitor. The delay between the beginning of arm cuff inflation and EEG arousal (when present) was noted. The delay between cuff inflation and a 10 mmHg rise in diastolic blood pressure was also noted (whenever a rise occurred). Some measurements were not included in the analysis for one of three reasons: the patient was not asleep before cuff inflation; body movements and especially left arm movements were seen on video monitoring during measurement; cuff inflation occurred during disturbances of breathing, which are known to induce haemodynamic changes and EEG arousal [25]. Respiratory disturbances were defined as apnoea when cessation of respiratory flow was longer than 10s and as hypopnoea when a reduction in respiratory flow of more than 50% was associated with a desaturation greater than 4%. A total of 85 ABP measurements from the nine patients were analysed.

Control measurements

The same number of periods (85) of the same duration and during the same stages of sleep as the periods of arm cuff inflation were selected at random in the nine patients. Heart rate and blood pressure were averaged using the same method as for the inflation measurements. On the same tracings, any occurrence of arousal was noted.

Statistical analysis

All data were computed and analysed with the SPSS-PC software [26]. Control periods were compared by analysis of variance (ANOVA) for blood pressure and arousals. Paired t-tests compared stages of sleep before and after arm cuff inflation, stages of sleep before and after Finapres resumption, as well as time delays for EEG arousal and rise in blood pressure. The number of EEG arousals was compared between sleep stages by one-way ANOVA. Linear regression was used to study the link between EEG arousal duration and increase in systolic blood pressure. The effect of cuff inflation on changes in blood pressure and heart rate was tested by ANOVA with the following factors: presence of hypertension, significant respiratory disturbance during sleep (apnoea-hypopnoea index > 10 per hour of sleep), sleep stage at the onset of arousal and the occurrence of arousal. Results are expressed as means ± SD.

RESULTS

Sleep efficiency and amounts of sleep stages varied from patient to patient, but total sleep time was 307 ± 12 min (range 257–364 min) (Table 1). Four patients had significant sleep apnoea (Table 1) with mean apnoea-hypopnoea index 46 ± 14 (range 25–73) per hour of sleep. Among these apnoeic patients, two were hypertensive and the other two were normotensive.

Effects of ABP on sleep

Statistical analysis showed significant sleep stage shifts (P = 0.01) associated with 25% of cuff inflations. Slow-wave sleep (stages 3 and 4) was significantly reduced, as was light sleep (stage 1), which shifted to wakefulness (P < 0.05). Awakenings (defined as arousals longer than 60s) occurred in 14% of measurements and EEG arousals lasting less than 60s were observed in 50% of measurements. Overall, 64% of cuff inflations were associated with some degree of arousal. Most EEG arousals were brief and lasted between 10 and 20s, as shown in Fig. 2. Arousals occurred in all sleep stages, including REM (Fig. 2). The presence of sleep apnoea syndrome, observed in four patients, had no significant effect on sleep stage shifts with cuff inflation. During control measurements only seven EEG arousals in 85 measurements were observed and no awakening occurred (P < 0.001, control versus inflation).

Effect of ABP on blood pressure and heart rate

Values of SBP, DBP and heart rate before and during cuff inflation are shown in Table 2.
cases, but there was no awakening and no significant stage shift during reinflation of the finger cuff after 5 min deactivation.

DISCUSSION

Our study shows that ABP measurement during sleep disturbs sleep architecture, causing numerous EEG arousals, and induces a significant rise in blood pressure at the time of systolic pressure measurement by the monitor. Awakenings causing body movements, and in particular arm movements, were discarded as a result of the artefact on the Finapres signal. Consequently, the most significant arousals and rises in blood pressure were probably not taken into account in the analysis. Our subjects were patients usually studied in a sleep laboratory for sleep-related breathing disorders, i.e. middle-aged patients with snoring complaint. Four of our patients were being treated for hypertension. Indeed, these patients have a high prevalence of hypertension [27]. Conversely, a high prevalence of sleep-disordered breathing has been suggested in patients with hypertension [28]. In these patients, ABP is widely used for evaluation of antihypertensive treatment or prognosis of hypertension. These patients reacted similarly to the healthy subjects studied by Davies et al. [20] using the same methods.

Effects of ABP on sleep

Although we used a silent monitor (Nippon Colin), we observed the same proportion of arousals (64%) as reported in other studies using noisier devices (67% [15], 61% [20]). This suggests that the tactile stimulus might be the most important sleep-disruptive factor. We observed the same percentage of arousals in all stages of sleep, including slow-wave sleep, for which the arousal threshold is considered to be higher [29]. The same finding was observed by Schwan and Eriksson [15], and may be taken as evidence that cuff inflation produced a fairly strong stimulus for arousal. Even when EEG arousal was brief and therefore did not wake the patient up, normal sleep architecture was interrupted by lighter sleep replacing slow-wave sleep. The evaluation of this disturbance of sleep architecture needs a precise analysis of EEG tracings, which has not been done in previous studies [14, 15].

Effects of ABP on blood pressure and heart rate

We chose to study 10-s averages for blood pressure and heart rate values in order to minimize any artefact that could alter a single value. Although the actual maximum rise in blood pressure may have been underestimated by this method, it remained highly significant. There was a large inter-individual variability in the blood pressure response to stimulus, from no rise in any of the measurements to a 57 mmHg rise. Our overall results are contrary to
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Fig. 3. Distribution of changes in SBP (a) and DBP (b) with cuff inflation (hatched bars) compared with changes during control measurements randomly chosen in the same sleep stages (full line).

Fig. 4. Effect of cuff inflation on SBP measured by Finapres (P1, ■) according to sleep stage and to systolic pressure measured before cuff inflation (P0, ●). SBP decreases with sleep stages from stage I to stages 3+4 and rises in REM (P0). In all sleep stages cuff inflation (P1) significantly increases SBP compared with P0 (*P < 0.01, **P < 0.001).

Relationship between arousal and blood pressure rise

In 20% of measurements, the blood pressure rise occurred without any noticeable modification in EEG pattern. Indeed, EEG arousal is not necessary for autonomic peripheral activation. This has been called the 'orienting reflex' by Johnson and Lubin [30]. It is an autonomic activation without EEG arousal caused by an external stimulus. It may be the result of brainstem activation without cortical arousal. In contrast, in 9% of measurements we observed an EEG arousal without blood pressure rise, usually during REM sleep. When both EEG arousal and blood pressure rise occurred, EEG modification preceded blood pressure rise and the longest EEG arousals were associated with the higher rises in blood pressure. These results suggest that EEG arousal and blood pressure elevation are not causally related but may both be triggered by the external stimulus via brainstem activation. In 16% of measurements, we observed a fall in blood pressure associated with cuff inflation. This fall was usually moderate and fell within the limits of normal blood pressure respiratory variability (around 5 mmHg). However, in one case, there was a 20 mmHg fall in SBP associated with a shift of sleep stage towards tonic REM sleep. There is no obvious explanation for this finding.
Practical conclusion

This study confirms that ABP measurements recorded during sleep in a sleep laboratory, especially the systolic values, should be interpreted with caution in patients suspected of suffering from sleep-disordered breathing, and means to evaluate sleep disturbances due to the ABP devices should be implemented. Individual responses to the stimulus are variable, but it is noteworthy that a pressure response was also observed in patients receiving antihypertensive treatment. This variability could be explained by the fact that some of the patients displayed sleep apnoea syndrome and/or systemic hypertension.

REFERENCES

Hypertension 1983; 5: 264-69.