Reproducibility of ambulatory blood pressure measurement in patients with coronary heart disease

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Summary

Ambulatory blood pressure monitoring (ABPM) is a useful tool to establish the diagnosis of hypertension as well as for the monitoring of the response to antihypertensive therapy. We aimed to assess the reproducibility of the circadian blood pressure (BP) pattern (24 h, daytime and night-time BP and heart rate HR), the categorisation of individuals as dippers and non-dippers and the morning BP peak in three consecutive ABPM recordings obtained in patients with coronary artery disease.

Methods: We performed a retrospective analysis of the reproducibility of 3 repeated ABPM obtained in 49 patients with coronary artery disease (age: 61.4 ± 7.9 years, 41 males, 36 hypertensives) enrolled in two previous studies using Tracker NIBP2 devices (Delmar, Del Mar Reynolds Medical, Hertford, UK). The standard interval of measurement was 20 minutes during the day and 45 minutes at night. The patients underwent ABPM on three separate days at intervals two to six weeks apart, on a typical week day with normal daily activity. All patients had a history of coronary artery disease and stable cardiovascular medication during the period of the three measurements.

Results: Using analysis of variance for repeated measurement (ANOVA, Pillai's trace test) and by Pearson's correlation coefficient there was no significant variance in 24–hour, day and night BP and HR as well as morning BP peak. The categorisation of the subjects as dippers and non-dippers was also highly reproducible ($p = 0.852$; ns).

Conclusions: Our data confirm that in patients with coronary artery disease the 24–hour recording of the circadian blood pressure patterns using Tracker NIBP2 devices is highly reproducible.

Key words: ABPM; ambulatory blood pressure measurement; coronary heart disease; reproducibility

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Introduction

The non-invasive ambulatory blood pressure monitoring (ABPM) was introduced in the seventies of the last century. These devices allow repetitive blood pressure (BP) and heart rate (HR) measurements over a period of 24 or 48 hours in an outpatient setting. Data in large cohorts have been accumulated to establish normal values as well as the importance of this diagnostic method. Based on such studies, it has been shown, that ABPM is very helpful for the diagnosis of hypertension as well as for monitoring the response to antihypertensive therapy [1–4]. In addition, ABPM is a very useful tool for a comprehensive evaluation of BP in special conditions such as secondary hypertension, hypotension, in elderly patients, children and pregnant women. The association with target organ damage such as left ventricular hypertrophy, proteinuria and cerebral microvascular lesions is more reliable with ABPM measurements than clinical or home BP measurements [5].

Further analysis revealed, that the 24–hour pattern of blood pressure is of importance, since not only the average of all 24–hour blood pressure values obtained, but also the fall of blood pressure during sleep as well as the rise of blood pressure in the early morning hours are of prognostic significance, therefore, it is of interest to get reliable data from ABPM measurements.

Normally, BP falls during sleep, due to the reduction of sympathetic nerve activity and a paralleled increase vagal tone [6]. A fall of greater than 10% of the daytime mean BP defines a dipper. Furthermore, a non-dipper profile in men correlates with the severity of coronary artery disease [7]. Non-dippers also show an increased prevalence of silent cerebrovascular disease, ischaemic and haemorrhagic stroke [8, 9]. Using ABPM it has been shown that a morning rise of ≥135/85 mm Hg is associated with increased cardiovascular mortality [10–12].

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The degree of reproducibility of blood pressure measurement even under standardised conditions has been discussed repeatedly. ABPM measurements seem to be more reliable than those obtained with an office sphygmomanometer [13, 14]. Data further indicate that the reproducibility of ABPM in normotensive patients is better than in hypertensives [14–18]. However, several authors questioned the reproducibility of ABPM. Doubts have been raised on the reproducibility of the categorisation of patients as dippers or non-dippers as well as regards the reproducibility of repeated measurements [14, 16, 18–22]. There are many factors affecting the reproducibility of ABPM measurements such as proper calibration, quality of the BP device as well as correct position, size of the cuff and an accurate arm position during the measurement [23]. Thijs et al. compared the different time-intervals of ABPM readings and could demonstrate that a minimum of two measurements per hour is required to guarantee an accurate assessment of the 24–hour standard deviation (SD) [24]. To get reliable data, measurements during the day should be done every 15 to 20 minutes and at night ordinarily every 30 to 60 minutes [1, 25, 26]. Although, several trials have shown that ABPM is very useful for risk assessment of hypertension, it seems not yet commonly used by most hospitals or doctors [27]. But we need further studies to verify the compliance with guidelines and using ABPM due to guidelines. This may be related to the fact that recent guidelines do not recommend ABPM as a routine examination in all patients in whom a diagnosis of hypertension is suspected. However, it is considered useful in patients with a certain variability of office or home blood pressure, with possible white coat hypertension, with masked hypertension, with a resistant hypertension and with signs/symptoms of hypotension during an antihypertensive treatment [3, 4].

In our previous studies [28, 29] we used ABPM to assess changes in blood pressure with drugs or food additives with uncertain effects on blood pressure. Here we used these datasets to study the reproducibility of ABPM.

Methods

We performed a retrospective analysis of the reproducibility of 3 repeated ABPM measurements obtained in 49 patients with coronary heart disease (age: 61.4 ± 7.9 years, 41 males, 36 hypertensive patients) enrolled in two previous studies performed at our institution between 2006 and 2010. All the ABPM measurements were performed using a validated device (Tracker NIBP2, Delmar, Del Mar Reynolds Medical, Hertford, UK). [30] In these previous studies approved by our institutional ethical committee all patients have granted their informed consent [28, 29]. For this retrospective analysis pseudonymised data of these two previous pharmaceutical studies were used.

To ensure that in our study and in the original studies there is no influence of drug-intake, studies were performed as a randomised crossover study. Between the first and the second part there was a washout period of two weeks in-between.

Patient selection

All patients have had a history of coronary artery disease (CAD) and stable cardiovascular medications during the period of the study. There were 36 patients with a history of arterial hypertension. The mean age of our patients was 61.4 years (range of 42 to 74 years). Males were 41 of 49 patients (84%). The patients had been recruited at the Department of Cardiology, University Heart Centre, University Hospital Zurich, Switzerland. Patients were included with CAD (documented by coronary angiography, nuclear imaging, or a positive bicycle test) on stable cardiovascular medication for at least one month. The age of inclusion criteria for patients was between 18 to 80 years.

Table 1

Pharmaceutical treatment and characteristics of the patients. ACE-I = Angiotensin-Inhibitor, AngII = Angiotensin II-Inhibitor, HCT = Hydrochlorothiazide, Ca-Antagonist = Calcium-Antagonist, BMI = Body mass index, MI = Myocardial infarction, PAD = Peripheral artery disease.

Pharmaceutical treatment, (N = 49)	
Aspirin; no. (%)	48 (97.96%)
Clopidogrel; no. (%)	14 (28.57%)
ACE-I/AngII-I; no. (%)	41 (83.67%)
HCT; no. (%)	13 (26.53%)
Other diuretic; no. (%)	2 (4.08%)
Beta blocker; no. (%)	28 (57.14%)
Ca-Antagonist; no. (%)	12 (24.49%)
Alpha blocker; no. (%)	2 (4.08%)
Antilipidemic; no. (%)	47 (95.92%)
Demographic characteristics (N = 49)	
Characteristic	
Age (year); mean ± SD	61.4 ± 7.9
BMI (kg/m2); mean ± SD	28.2 ± 4.5
Diabetes; no. (%)	7 (14.3%)
History of MI/PAD; no. (%)	41 (83.7%)
Dyslipidaemia; no. (%)	45 (91.8%)
History of hypertension; no. (%)	36 (73.5%)
Active smoker; no. (%)	1 (2%)
History of smoking; no. (%)	30 (61.2%)
Sex, male; no. (%)	41 male (83.70%)
Numbers of antihypertensive medication (N = 49)	Mean
0	2 (4.08%)
1	13 (26.53%)
2	21 (42.86%)
3	10 (20.41%)
4	3 (6.12%)

Exclusion criteria were acute myocardial infarction, unstable angina, stroke or percutaneous coronary intervention or surgical revascularisation procedure within three months before study entry; left ventricular ejection fraction <50%; use of other analgesics (platelet inhibition therapy with aspirin 100 mg/d was continued); chronic pain; smoking, alcohol, or substance abuse; uncontrolled BP despite adequate therapy (>160/100 mm Hg); renal failure (serum creatinine >200 µmol/l); liver disease (alanine aminotransferase or aspartate aminotransferase >100 IU); acute hepatitis; hyperbilirubinemia; concomitant therapy with oral anticoagulants, phenobarbital, phenytoin, carbamazepine, isonicotinic acid, chloramphenicol, chlorzoxazone, zidovudine, and salicylamide; long-term use of nitrates; insulin-dependent diabetes mellitus; anaemia (haemoglobin <10 g/dl); systemic inflammatory diseases (e.g., rheumatoid arthritis, Crohn's Disease); known human immunodeficiency virus infection or active virus – hepatitis; pregnancy or breast-feeding, women with childbearing potential without adequate contraception; malignancy (unless healed or in remission >5 years); recipient of any major organ transplant (e.g., lung, liver, heart) or renal replacement therapy; and participation in another study within the last month.

Patients were on stable cardiovascular medication and were not allowed to change the therapy (in particular, no anti-inflammatory and pain-relieving drugs (table 1).

ABPM measurement

ABPM measurements were obtained over 24 hours with the Tracker NIBP 2 (Delmar, Del Mar Reynolds Medical, Hertford, UK). The standard interval of measurement was 20 minutes during the day and 45 minutes at night as recommended by the guidelines [4, 31]. The patients underwent ABPM on three separate days two to six weeks apart. All measurements were performed on a typical weekday without planned special activities. The inflating cuff was worn on the non-dominant arm and the patients were asked to keep their arm calm whenever the cuff was inflating and to avoid excessive physical exertion during monitoring. Our patients were provided with an activity diary to record the times of daily activities, of going to bed and getting up. The night-and daytime periods were determined individually according to the information obtained by each patient. ABPM analysis was done using a special computer program (Cardio Navigator) providing mean daytime, night and 24 hours BP as well as HR and morning peak BP. In the measurements of morning peak there was in one patient once an early termination of the ABPM measurement (at the time of V2), therefore we excluded this recording for the statistical calculation.

Office blood pressure measurements

The office BP was measured at every visit using an automated BP monitor (Omron, Healthcare Co., Ltd.,

Table 2
Pillai correlation coefficients and P-values. Repeated measurements show no significant differences in ABPM. Only in clinical measurements there is a significant difference in DBP.

Variable	First ABPM mean ± SD [95%-Confidence interval]	Second ABPM mean ± SD [95%-Confidence interval]	Third ABPM mean ± SD [95%-Confidence interval]	Repeated measures ANOVA Pillai (significance)
24_H_SBP	123.2 ± 11.3; [120.0; 126.4]	123.0 ± 11.2; [119.8; 126.2]	125.3 ± 11.3; [122.0; 128.5]	0.063 (ns)
24_H_DBP	73.6 ± 6.9; [71.6; 75.5]	73.7 ± 7.4; [71.5; 75.8]	74.6 ± 6.8; [72.7; 76.6]	0.177 (ns)
24_H-MBP	84.4 ± 7.5; [82.3; 86.6]	84.3 ± 8.1; [82.0; 86.6]	85.7 ± 7.4; [83.6; 87.7]	0.084 (ns)
24_H-HR	68.6 ± 9.6; [65.9; 71.4]	69.7 ± 9.5; [67.0; 72.4]	69.5 ± 8.9; [67.0; 72.1]	0.476 (ns)
Day_SBP	126.2 ± 11.8; [122.9; 129.6]	125.5 ± 12.1; [122.0; 129.0]	127.5 ± 11.6; [124.2; 130.9]	0.189 (ns)
Day_DBP	75.9 ± 7.4; [73.8; 78.0]	75.6 ± 8.0; [73.3; 77.9]	76.4 ± 7.1; [74.3; 78.4]	0.516 (ns)
Day_MBP	86.8 ± 8,0; [84.5; 89.1]	86.4 ± 8.7; [83.9; 88.9]	88.4 ± 10.0; [85.5; 91.3]	0.224 (ns)
Day_HR	70.8 ± 9.5; [68.1; 73.5]	72.7 ± 9.7; [67.0; 75.5]	71.4 ± 9.3; [68.7; 74.0]	0.209 (ns)
Night_SBP	114.9 ± 11.2; [11.7; 118.1]	114.3 ± 9.9; [11.5; 117.1]	114.8 ± 19.6; [109.2; 120.4]	0.849 (ns)
Night_DBP	66.4 ± 7.8; [64.1; 68.6]	66.3 ± 7.0; [64.3; 68.3]	66.1 ± 10.8; [63.0; 69.2]	0.985 (ns)
Night_MBP	77.0 ± 8.5; [74.6; 79.5]	76.9 ± 7.3; [74.8; 79.0]	79.1 ± 9.8; [76.3; 81.9]	0.161 (ns)
Night_HR	61.6 ± 8.4; [59.1; 64.0]	62.4 ± 9.4; [59.7; 65.1]	63.5 ± 9.4; [60.7; 66.2]	0.086 (ns)
Mean_clin_SBP	132.4 ± 13.4; [128.6; 136.3]	131.3 ± 11.5; [128.0; 134.6]	130.1 ± 12.7; [126.5; 133.8]	0.412 (ns)
Mean_clin_DBP	80.0 ± 8.1; [77.6; 82.3]	81.4 ± 8.4; [79.0; 83.8]	78.9 ± 7.4; [126.5; 133.8]	0.014 (s)
Mean_clin_HR	59.2 ± 9.3; [56.5; 61.9]	60.1 ± 9.9; [57.3; 63.0]	59.4 ± 8.1; [57.0; 61.7]	0.678 (ns)
Dipper/ Non-Dipper				0.852 (ns)
MorningPeak_SBP	130.7 ± 19.6; [125.0; 136.4]	129.8 ± 16.0; [125.1; 134.4]	129.9 ± 17.2; [124.9; 134.9]	0.925 (ns)
MorningPeak_DBP	77.3 ± 10.3; [74.4; 80.3]	78.4 ± 11.9; [75.0; 81.9]	79.2 ± 10.6; [76.1; 82.3]	0.511 (ns)

ABPM = Ambulatory blood pressure monitoring, ns= not significant, s= significant, SD = Standard deviation of all measurements, SBP = systolic blood pressure, DBP= diastolic blood pressure, MBP = mean blood pressure, HR = heart rate, clin = clinical measurements

Table 3
Fisher Transformation with confidence interval shows strong correlation of each value of Pearson's correlation. As you see in the table there is a certain stability of each individual.

Fisher Transformation (Confidence interval)		
Correlation (Pearson)	**Mean_24H_SBP_2**	**Mean_24H_SBP_3**
Mean_24H_SBP_1 *1 *2	**0.837** [0.73; 0.91] <0.000	**0.818** [0.70; 0.89] <0.000
Mean_24H_SBP_2		**0.796** [0.66; 0.88] <0.000
Mean_24H_DBP_1	**0.856** [0.76; 0.92] <0.000	**0.816** [0.69; 0.89] <0.000
Mean_24H_DBP_2		**0.814** [0.69; 0.89] <0.000
Mean_24H_MBP_1	**0.850** [0.75; 0.91] <0.000	**0.812** [0.69; 0.89] <0.000
Mean_24H_MBP_2		**0.805** [0.68; 0.89] <0.000
Mean_Day_SBP_1	**0.839** [0.73; 0.91] <0.000	**0.812** [0.69; 0.89] <0.000
Mean_Day_SBP_2		**0.793** [0.66; 0.88] <0.000
Mean_Day_DBP_1	**0.830** [0.72; 0.90] <0.000	**0.801** [0.67; 0.88] <0.000
Mean_Day_DBP_2		**0.813** [0.69; 0.89] <0.000
Mean_Day_MBP_1	**0.844** [0.74; 0.91] <0.000	**0.658** [0.46; 0.79] <0.000
Mean_Day_MBP_2		**0.658** [0.46; 0.79] <0.000
Mean_Night_SBP_1	**0.770** [0.62; 0.86] <0.000	**0.443** [0.18; 0.64] <0.001
Mean_Night_SBP_2		**0.440** [0.18; 0.64] 0.002
Mean_Night_DBP_1	**0.763** [0.61; 0.86] <0.000	**0.481** [0.23; 0.67] <0.000
Mean_Night_DBP_2		**0.442** [0.18; 0.64] 0.001
Mean_Night_MBP_1	**0.752** [0.60; 0.85] <0.000	**0.598** [0.38; 0.75] <0.000
Mean_Night_MBP_2		**0.560** [0.33; 0.73] <0.000
Morning Peak_SBP_1	**0.600** [0.38; 0.75] <0.000	**0.438** [0.18; 0.64] <0.002
Morning Peak_SBP_2		**0.566** [0.34; 0.73] <0.000
Morning Peak_DBP_1	**0.590** [0.37; 0.75] <0.000	**0.444** [0.19; 0.64] 0.002
Morning Peak_DBP_2		**0.664** [0.47; 0.80] <0.000
Mean_HF_Day_1	**0.682** [0.50; 0.81] <0.000	**0.813** [0.69; 0.89] <0.000
Mean_HF_Day_2		**0.770** [0.62; 0.86] >0.000
Mean_HF_Night_1	**0.861** [0.77; 0.92] <0.000	**0.795** [0.66; 0.88] <0.000
Mean_HF_Night_2		**0.828** [0.71; 0.90] <0.000
Mean_HF_24h_1	**0.799** [0.67; 0.88] <0.000	**0.776** [0.63; 0.87] <0.000
Mean_HF_24h_2		**0.901** [0.83; 0.94] <0.000

*1) Fisher z-Transformation r (Confidenc e interval)

*2) Pearson-Correlation p

Table 4
Morning peak (MP; synonym: morning rise, morning surge) of ABPM (categorisation in ≥ or <135 mm Hg). The definition of a normal morning peak is the first blood pressure after awakening <135 mm Hg. An exaggerated morning peak (>135 mm Hg) is associated with an increased cardiovascular mortality [2–4].
For the measurements of morning peak there was in one patient once an early termination of the ABPM measurement (at the time of V2), therefore we excluded this recording for the statistical calculation. The categorisation of exaggerated normal morning peak (BP ≥135 mm Hg) was stable in 28 of 48 patients. In 20 patients there was a change in the classification in categories normal/exaggerated of the morning peak.

Exaggerated morning peak (MP) ≥135 mm Hg, N = 48	Numbers of pat. [range]
V1	18 [135–198 mm Hg]
V2	19 [135–164 mm Hg]
V3	15 [135–174 mm Hg]
MP in 3 measurements:	
Classification in normal and exaggerated MP was stable in 3 measurements in 28 pat.	
V1 = first measurement, V2 = second measurement; V3 = third measurement.	

Kyoto, Japan; Elite Plus, HEM-7301–ITKE, CE 0197). According to guidelines, three readings 1–2 minutes apart were performed in a sitting position after at least five minutes rest and then the average of the last two measurements was used.

Statistical analysis

Blood pressure values were expressed as mean ± standard deviation (SD).

For the evaluation of the reproducibility of the variables measured during three separate recordings analysis of variance (ANOVA) for repeated measurements were used. For correlation analysis, Fisher-z-Transformation and then Pearson's correlation coefficients were applied. The multivariate analysis was completed using the Pillai-test. The statistical significance was considered at a value of $p < 0.05$.

Database management and statistical analysis were performed using SPSS software (SPSS Inc., Chicago, IL, version 20).

Results

Overall reproducibility

In the present study, ABPM at three different occasions; days, two to six weeks apart in a group of 49 patients with coronary artery disease and a stable medication regimen, measurements were highly reproducible. In some patients, the 24–hour as well as daytime-BP and night-BP showed hypertensive values. There was no significant variance in 24–hour, day and night BP and heart rate (HR) as well as in the morning peak (table 2). Fisher Transformation with confidence interval showed also a strong correlation of each value of Pearson's correlation (table 2). As can be seen in table 3, individual values were stable.

Morning peak

An exaggerated morning peak of over 135 mm Hg in the first measurement was noted in 18 patients (with a range of 135 to 198 mm Hg), while in the second measurement this was noted in 19 patients (with a range of 135 to 164 mm Hg) and in the third measurement in 15 patients (with a range of 135 to 174 mm Hg). The stability of categorisation in exaggerated and normotensive morning peak was stable in 28 patients of the 48 included (table 4).

Dippers and non-dippers

Of the 49 patients included, 9 patients were dippers in all three recordings. In 24 patients the categorisation of dipper or non-dipper, respectively was stable in all 3

Table 5
Categorisation as hypertensive or normotensive due to each measurement of ABMP. In most individuals the categorisation was stable, but not in everyone. However, one has to take into account that in the most cases the changes in categorisation were due to changes in blood pressure within a few mmHg around the ranges of the categorization in hypertensive/normotensive. The classification in categories hypertensive/normotensive values is however of clinical relevance due to the fact that it impacts on the decision for medical treatment.

Categorisation due to ABPM-values, N = 49				
	Categorisation as hypertensive	**Stable hypertensive, n**	**Stable normotensive, n**	**Changes, n**
24–H-SBP	SBP >130 mm Hg	9	29	11
24–H-DBP	DBP >80	4	35	10
Day-SBP	SBP >135 mm Hg	7	34	8
Day-DBP	DBP >85	1	39	9
Night-SBP	SBP >120 mm Hg	6	32	11
Night-DBP	DBP >70	6	28	15
N = number of all patients, n = number, BP = blood pressure, 24–h = 24 hours, SBP = systolic blood pressure, DBP = diastolic blood pressure				

measurements. The classification in dipper and non-dipper was highly reproducible (p = 0.852; ns; Pillai, repeated measures ANOVA) (table 2).

Office blood pressure

In each visit we performed office BP measurements. Only the clinical diastolic blood pressure differed statistically significantly. The variance of clinical systolic blood pressure (clin_SBP) and clinical heart rate (clin_HR) was statistically not significant (p-value 0.412 and p-value 0.678) (table 2).

Discussion

This retrospective study demonstrates a high reproducibility of the circadian blood pressure patterns in 24–hour recordings in patients with coronary artery disease under stable pharmacological therapy. To our knowledge this is the first study evaluating the reproducibility of repeated ABPM in patients with coronary heart disease and stable medication. Our study demonstrates that in this special population the results of 24–hour blood pressure measurements are highly reproducible. This finding is of relevance for the use of ABPM to either diagnose hypertension in these patients as well as to study drug effects in studies or in treatment patients.

It seems that in our study the reliability of the clinical blood pressure measurement was poorer than the ABPM measurements. One explanation for the poor reproducibility of office blood pressure measurements could be that the daytime visits at our outpatient clinic was not standardised to a fixed time of the day. Furthermore, there is a biological variability of blood pressure due to physical activities and stressful situations [32]. As described in the study from van der Wel et al. (2011) the results of office blood pressure are more reliable, if office measurements are performed every 5 minutes over a period of 30 minutes. Under these conditions, office blood pressure may be equivalent to ABPM [33]. In our study, office blood pressure measurements may also differ from ABPM, because different devices were used under both circumstances. The poorer reproducibility of diastolic office blood pressure measurements in our study was statistically significant, but the change from baseline was on average within 2.5 mm Hg which is clinically not very relevant. Similarly, in a study by Jula et al. blood pressure variation between four office measurements was small, but statistically significant [34].

Previously, similar data have been reported in healthy normotensive adults as well as in untreated and treated patients with arterial hypertension [32, 35–42]. In accordance with our results, many studies have shown that ambulatory BP values are more reproducible than office BP values [13, 43–46]. In contrast to the present findings, however, there were also studies showing a poor reproducibility of ABPM recordings [16, 21]. Gerin et al. described in 1993 a lack of standardisation of activities of each patient from one session to another as a reason for poorer reproducibility of ambulatory blood pressure measurements [16, 22, 46]. For example, in the study of Hernandez-del Rey the night-time rest period was defined (between 0100 and 0500h) [32]. Unlike other studies, we have defined the daytime and nighttime periods on the basis of the diary protocols provided by our patients, an approach that may have contributed to the high reproducibility as described by Weston et al. in 1996 the reproducibility was poor when data were analysed using standard definitions of day and night [47].

One explanation for the excellent reproducibility could be the fact that most patients had normotensive BP values and a stable medication regimen during the observation period of this study. Indeed, Musso described in 1996, that work, physical activity and untreated hypertensive blood pressure values reduce the reproducibility reliability of ABMP measurements [14]. Furthermore, in this study all patients were instructed to avoid any movements of the arm, while measurements were made which may have also contributed to the excellent reproducibility. Finally, patients with coronary artery disease are likely to show atherosclerosis in other vascular beds. As such carotid atherosclerosis may impair baroreceptor function. Therefore regulation and variation of blood pressure may also be affected. The major change in patients with atherosclerosis is an increased ratio of nitrate and cyclic GMP, which translates into increased oxidative inactivation of NO in these kinds of patients. A disturbed NO build-up and activity of NO may contribute to blood pressure alterations in cardiovascular disease [48]. In table three you can see a poorer correlation of the night time data. As you would already expect there is a stronger correlation in the night time data as opposed to the daytime data due to the lesser influence of daily activity and stressful situations, but it has already been described in a previous study of Cuspidi et al. (2011) that potential factors, for example, during bed-rest-period and having sufficient BP records at night could have an influence of the reproducibility of data. In our study we did not define the minimum and maximum time of bed-rest-period. Therefore, it could be one explanation of the poorer correlation of the night-BP data in contrast to the daily-BP data [49].

Some limitations of this study should be acknowledged: First, the data of this study were obtained in a retrospective manner and the sample size was small. However, as the latter aspect would work against a high reproducibility, the results obtained appear the more valid.

In conclusion, our study confirms that ABPM measurements are well reproducible.

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