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Original Paper

Cuff-Based Oscillometric Central and Brachial Blood Pressures Obtained Through ABPM are Similarly Associated with Renal Organ Damage in Arterial Hypertension

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Key Words
Aortic blood pressure • Albuminuria • Glomerular filtration rate

Abstract
Background/Aims: Central blood pressure (BP) has been suggested to be a better estimator of hypertension-associated risks. We aimed to evaluate the association of 24-hour central BP, in comparison with 24-hour peripheral BP, with the presence of renal organ damage in hypertensive patients. ***Methods:*** Brachial and central (calculated by an oscillometric system through brachial pulse wave analysis) office BP and ambulatory BP monitoring (ABPM) data and aortic pulse wave velocity (PWV) were measured in 208 hypertensive patients. Renal organ damage was evaluated by means of the albumin to creatinine ratio and the estimated glomerular filtration rate. ***Results:*** Fifty-four patients (25.9%) were affected by renal organ damage, displaying either microalbuminuria (urinary albumin excretion ≥30 mg/g creatinine) or an estimated glomerular filtration rate (eGFR) <60 ml/min/1.73 m^2. Compared to those without renal abnormalities, hypertensive patients with kidney damage had higher values of office brachial systolic BP (SBP) and pulse pressure (PP), and 24-h, daytime, and nighttime central and brachial SBP and PP. They also had a blunted nocturnal decrease in both central and brachial BP, and higher values of aortic PWV. After adjustment for age, gender, and antihypertensive treatment, only ABPM-derived BP estimates (both central and brachial)

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showed significant associations with the presence of renal damage. Odds ratios for central BP estimates were not significantly higher than those obtained for brachial BP. ***Conclusion:*** Compared with peripheral ABPM, cuff-based oscillometric central ABPM does not show a closer association with presence of renal organ damage in hypertensive patients. More studies, however, need to be done to better identify the role of central BP in clinical practice.

Introduction

Cardiovascular (CV) risk assessment is based on measurement of peripheral or brachial BP. Central or aortic BP, however, is expected to better reflect the hemodynamic load on target organs. Indeed, clinical studies suggest that central BP shows a closer relationship with target organ damage than does brachial BP [1–3]. Preclinical heart damage assessed by left ventricular mass and large artery damage assessed by intima media thickness and pulse wave velocity (PWV) appear to be closely associated with central systolic BP (SBP) and central pulse pressure (PP). Data on the relationship of central BP and renal organ damage are, however, conflicting [4–6]. It is widely known that 24-h ambulatory BP monitoring (ABPM) is a better predictor of target organ damage and CV risk than office BP [7]. ABPM provides a large number of BP measurements together with nighttime BP measurement, reflecting an important part of the hypertension-related load on the CV system. Technological advances in recent years have allowed the development of devices capable of estimating central 24-h ABPM noninvasively by the cuff-based oscillometric method (Mobil-O-Graph, IEM, Germany) [8, 9]. Estimation of 24-h central BP can improve CV risk assessment in arterial hypertension. Therefore, the aim of the present study was to evaluate the possible association between 24-h central hemodynamic BP indices and renal organ damage in hypertensive patients.

Materials and Methods

Study design

This was a cross-sectional study including 208 patients aged >18 years with a diagnosis of essential hypertension (mean age 57±12 years, 66% men) who were consecutively enrolled from five hypertension units at corresponding university hospitals in the metropolitan area of Barcelona, Spain. The study was approved by the local institutional ethics committees. Written informed consent was obtained from all participants.

Anthropometric and demographic characteristics, CV risk factors, and laboratory results were recorded at the inclusion visit for all participants. The following data were obtained: age, sex, weight, height, and family history of premature CV disease. Obesity was defined by a body mass index (BMI) ≥30 kg/m^2. Other recorded variables were smoking habit, duration of hypertension, antihypertensive treatment prescribed, and previous history of CV disease (including heart failure, coronary heart disease, cerebrovascular disease, and symptomatic ischemic peripheral vascular disease). Diabetes mellitus was diagnosed using the medical history if the patient was under antidiabetic treatment or by two or more fasting plasma glucose determinations ≥7.0 mmol/L (126 mg/dL). Dyslipidemia was considered to be present if patients were being treated with lipid-lowering drugs and/or total cholesterol was >5 mmol/L (190 mg/dL), low-density lipoprotein cholesterol was >3.0 mmol/L (115 mg/dL), high-density lipoprotein cholesterol was <1.0 mmol/L (40 mg/dL) (men) or <1.2 mmol/L (46 mg/dL) (women), or triglycerides were >1.7 mmol/L (150 mg/dL).

BP measurements

All BP measurements were performed by means of a noninvasive automated oscillometric device (Mobil-O-Graph PWV, IEM, Stolberg, Germany), validated for brachial BP measurement, according to the European Society of Hypertension International protocol [9]. The monitor was placed on a working day, starting between 08:00 AM and 10:00 AM. After 5-min rest, BP was measured 4 times consecutively at

1-min intervals. The mean of these measurements served as the office BP. Thereafter, BP was measured automatically at 20-min intervals throughout both the awake and the asleep period, as defined in the patient's dairy. Inclusion of subjects in the study was contingent upon acquisition of recordings of good technical quality (at least 80% valid readings). If this criterion was not met, ABPM was repeated in one week.

Assessment of renal organ damage

Serum creatinine was measured by an enzymatic modified Jaffe reaction (CREA; Roche Diagnostics), consistent with the current recommendations for standardizing serum creatinine measurement. Urinary albumin excretion (measured by turbidimetry in local laboratories according to current recommended standards) was determined as the average of the ratio of albumin to creatinine in two spot first-morning void urine samples obtained on separate days. The eGFR was calculated using the Chronic Kidney Disease–Epidemiology Collaborative equation (CKD-EPI) [10].

Kidney disease was considered present if the patient had a urinary albumin excretion ≥30 mg/g creatinine and/or an eGFR <60 ml/min/1.73 m^2.

Aortic pulse wave velocity

Aortic pulse wave velocity (aPWV) was measured by means of the Mobil-O-Graph device with the inbuilt ARCSolver method used for brachial and central 24-h BP assessment. The methodology for aPWV estimation has been previously reported and validated against invasive methods [11, 12]. A measurement was taken concomitant with each BP measurement and mean values for 24 h were computed.

Statistical analysis

Data are presented as mean ± standard deviation for normally distributed variables, median [interquartile range] for continuous variables that deviated from the normal distribution, or frequencies (%) for qualitative variables. Differences in clinical parameters and BP values between patients with or without renal damage were estimated by means of Student's t test, Mann-Whitney U-test, or Pearson's chi square test, as appropriate. The association of each BP estimate with the presence of renal damage was assessed by means of logistic regression analyses, with risk ratio calculation adjusted for age, gender, and the use of antihypertensive treatment. The SPSS for Windows version 18.0 software (SPSS Inc., Chicago, IL) was used for statistical analysis.

Results

Patients

Kidney data of hypertensive patients included in the study are shown in Table 1. Thirty-five patients (16.8%) had microalbuminuria and 28 (13.5%) had an eGFR <60 ml/min/1.73 m^2. A total of 54 patients (25.9%) were affected by renal organ damage, as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2. Clinical and demographic characteristics of the population with (n=54) and without (n=154) renal organ damage are shown in Table 2. As expected, hypertensive patients with kidney damage were older and had higher BMI and higher rates of diabetes, dyslipidemia, and previous CV disease. Accordingly, patients with renal organ damage had higher plasma levels of glucose, HbA1c, uric acid, and triglycerides and were receiving a higher number of antihypertensive drugs.

Table 1. Kidney data of hypertensive patients included in the study (n=208). Data are expressed as: mean ± standard deviation, median [interquartile range], or n (%) as applicable. Microalbuminuria: urinary albumin excretion ≥30 mg/g creatinine; eGFR: estimated glomerular filtration rate; UAE: urine albumin excretion

Creatinine (mg/dL)	0.96±0.28
eGFR (ml/min/1.73 m^2)	83.3±20.2
UAE (mg/g)	6.2 [3.5–17.7]
Microalbuminuria	35 (16.8%)
eGFR <60 ml/min/1.73 m^2	28 (13.5%)
Microalbuminuria or eGFR <60 ml/min/1.73 m^2	54 (25.9%)

Table 2. Clinical and demographic characteristics of hypertensive patients with or without renal organ damage (as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m²). Data are expressed as: mean ± standard deviation or median [interquartile range]. BMI: body mass index; CV: cardiovascular; eGFR: estimated glomerular filtration rate; HbA1c: glycated hemoglobin; OD: organ damage; UAE: urine albumin excretion

Variable	With renal OD n=54 (26%)	Without renal OD n=154 (74%)	p value
Male gender (%)	61.1	68.2	0.403
Age (years)	64.9±8.8	54.3±12.2	<0.001
BMI (kg/m²)	30.4±5.0	29.0±4.4	0.043
Family history of early CV event (%)	18.5	19.5	0.877
Smokers (%)	31.5	24.0	0.324
Diabetes (%)	29.6	16.2	0.046
Dyslipidemia (%)	81.5	55.2	0.001
Previous CV disease (%)	46.3	15.4	<0.001
Glucose (mg/dL)	115.5±28.5	105.9±28.7	0.034
HbA1c (%)	6.1±0.8	5.7±0.7	0.001
Uric acid (mg/dL)	6.3±1.7	5.6±1.4	0.001
Cholesterol, total (mg/dL)	177.2±35.1	181.2±36.7	0.493
Cholesterol, LDL (mg/dL)	101.7±30.1	107.1±33.7	0.274
Cholesterol, HDL (mg/dL)	52.0±17.8	54.8±18.0	0.329
Triglycerides (mg/dL)	142.3±56.6	110.4±54.6	<0.001
UAE (mg/g)	65 [11–177]	5 [3–10]	<0.001
eGFR (ml/min/1.73 m²)	64.9±22.1	89.8±14.4	<0.001
Currently on antihypertensive therapy (%)	98.1	87.0	0.018
Number of drugs	3.3±1.3	2.7±1.3	0.006

Table 3. Clinical and demographic characteristics of hypertensive patients based on the presence or absence of microalbuminuria or reduced renal function (eGFR <60 ml/min/1.73 m²). Data are expressed as mean ± standard deviation, median [interquartile range], or n (%), as applicable. BMI: body mass index; eGFR: estimated glomerular filtration rate; HbA1c: glycated hemoglobin; NA: not applicable; UAE: urine albumin excretion

Variable	Microalbuminuria			eGFR <60 ml/min/1.73 m²		
	With 35 (16.8%)	Without 167 (83.1%)	p value	With 28 (13.5%)	Without 179 (86.5%)	p value
Male gender (%)	65.7	66.5	0.932	57.0	67.8	0.287
Age (years)	63.5±8.6	55.9±12.4	0.001	66.0±8.8	55.7±12.2	<0.001
BMI (kg/m²)	30.6±5.2	29.2±4.5	0.106	29.5±4.3	29.3±4.7	0.821
Family history of early CV event (%)	17.1	20.4	0.817	25.0	18.3	0.440
Smokers (%)	34.3	24.6	0.271	32.1	25	0.667
Diabetes (%)	40.0	16.2	0.004	14.3	20.6	0.611
Dyslipidemia (%)	82.9	57.5	0.007	78.6	59.4	0.061
Previous CV disease (%)	42.9	19.8	0.008	57.1	18.3	<0.001
Glucose (mg/dL)	122.3±32.5	105.9±27.7	0.008	105.6±13.9	108.8±30.6	0.589
HbA1c (%)	6.3±0.8	5.7±0.7	<0.001	5.8±0.5	5.8±0.8	0.932
Uric acid (mg/dL)	6.3±1.8	5.7±1.5	0.033	6.9±1.9	5.6±1.4	<0.001
Cholesterol, total (mg/dL)	178.1±37.5	179.8±35.8	0.796	177.4±31.8	180.6±36.9	0.669
Cholesterol, LDL (mg/dL)	102.4±33.1	106.2±32.4	0.504	104.9±26.8	106.1±33.7	0.853
Cholesterol, HDL (mg/dL)	51.6±18.0	54.3±17.3	0.404	49.3±16.1	54.9±18.1	0.126
Triglycerides (mg/dL)	147.0±58.5	113.1±55.1	0.001	143.9±54.2	114.7±56.3	0.011
UAE (mg/g)	NA	NA	NA	11 [5-61]	6 [3.5-15.9]	<0.001
eGFR (ml/min/1.73 m²)	72.6±23.3	85.6±18.7	<0.001	NA	NA	NA
Currently on antihypertensive therapy (%)	97.1	89.2	0.207	100	88.3	0.085
Number of drugs	3.2±1.5	2.5±1.5	0.020	3.4±1.3	2.5±1.5	0.005

Clinical and demographic characteristics were further analyzed separately in patients with or without microalbuminuria and with or without reduced renal function. No distinctive features were directly associated with either microalbuminuria or reduced eGFR, with the exception of a poor metabolic profile, which differed between microalbuminuric and normoalbuminuric patients, but not between those with normal or reduced renal function (Table 3).

Table 4. Office and 24-h ambulatory brachial and central BP in patients with or without renal organ damage (as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2). Data are expressed as mean ± standard deviation. DBP: diastolic blood pressure; OD: organ damage; PP: pulse pressure; SBP: systolic blood pressure

BP	Brachial			Central		
	With renal OD (n=54)	Without renal OD (n=154)	p value	With renal OD (n=54)	Without renal OD (n=154)	p value
Office SBP	142.0±22.1	135.0±17.0	0.039	127.2±18.2	125.3±17.4	0.496
Office DBP	85.1±12.2	87.8±11.2	0.123	86.9±11.6	88.8±11.5	0.304
Office PP	55.4±17.0	46.8±13.3	0.002	40.2±10.8	36.9±14.2	0.126
24-h SBP	135.0±16.2	127.3±12.3	<0.001	122.8±15.0	117.9±12.5	0.020
24-h DBP	79.2±9.0	80.6±8.4	0.315	80.7±9.5	82.0±8.5	0.350
24-h PP	55.8±12.8	46.7±8.5	<0.001	41.0±8.9	35.3±6.4	<0.001
Daytime SBP	137.0±16.4	131.4±13.2	0.013	124.8±15.1	121.2±12.6	0.090
Daytime DBP	81.5±9.9	84.3±9.1	0.061	83.3±10.3	86.1±9.4	0.074
Daytime PP	55.6±12.6	47.0±8.9	<0.001	40.3±9.4	34.7±6.7	<0.001
Nighttime SBP	129.1±18.9	118.4±13.9	<0.001	117.9±18.2	110.8±15.5	0.006
Nighttime DBP	73.5±10.1	72.0±9.1	0.311	74.2±10.4	73.2±9.4	0.514
Nighttime PP	55.6±14.2	46.5±8.9	<0.001	42.6±10.1	37.0±7.8	<0.001
% nocturnal decrease in SBP	5.8±8.9	9.6±8.1	0.004	5.6±9.7	8.5±8.9	0.047
% nocturnal decrease in DBP	9.7±9.3	14.2±9.7	0.004	10.8±9.0	14.5±9.6	0.013

Office and 24-h ambulatory brachial and central BP

Office and 24-h ambulatory brachial and central BP are shown in Table 4. Patients with renal damage had higher SBP and PP values, both brachial and central. The magnitude of the difference was generally more prominent for brachial than for central BP. Furthermore, patients with kidney damage showed a blunted nocturnal decrease in both brachial and central BP. In line with the PP increase, patients with renal damage also had higher office and ambulatory daytime, nighttime, and 24-h aortic PWV (Table 5).

Table 5. Office and 24-h ambulatory aortic pulse wave velocity in patients with or without renal organ damage (as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2). Data are expressed as mean ± standard deviation. aPWV: aortic pulse wave velocity; OD: organ damage

	With renal OD	Without renal OD	p value
Office aPWV	9.7±1.7	8.1±1.6	<0.001
24-h aPWV	9.6±1.6	7.9±1.5	<0.001
Daytime aPWV	9.7±1.6	7.9±1.5	<0.001
Nighttime aPWV	9.4±1.7	7.7±1.5	<0.001

Table 6. Office and 24-h ambulatory brachial and central BP in hypertensive patients based on the presence or absence of microalbuminuria. Data are expressed as mean ± standard deviation. DBP: diastolic blood pressure; PP: pulse pressure; SBP: systolic blood pressure

BP	Brachial			Central		
	With (n=35)	Without (n=167)	p value	With (n=35)	Without (n=167)	p value
Office SBP	140.5±21.1	135.6±17.6	0.201	125.2±16.7	125.1±15.7	0.974
Office DBP	83.5±11.5	87.7±11.3	0.049	86.1±10.7	88.7±11.6	0.225
Office PP	55.3±18.5	47.4±13.0	0.020	39.0±10.2	36.8±11.3	0.286
24-h SBP	136.9±18.0	127.8±12.3	0.008	124.6±16.5	117.8±11.4	0.029
24-h DBP	80.2±8.8	80.3±8.5	0.930	81.7±9.5	81.7±8.6	0.994
24-h PP	56.7±14.4	47.6±8.7	0.001	41.6±9.8	35.8±6.6	0.002
Daytime SBP	138.4±18.2	131.6±13.1	0.042	125.8±16.7	121.1±11.8	0.125
Daytime DBP	82.4±9.9	83.9±9.3	0.412	84.0±10.2	85.6±9.5	0.376
Daytime PP	55.9±14.2	47.8±9.1	0.002	40.6±10.2	35.2±6.8	0.005
Nighttime SBP	132.0±20.8	119.1±13.7	0.001	120.7±19.7	110.8±13.3	0.008
Nighttime DBP	74.7±9.9	72.1±9.1	0.136	75.5±10.7	73.1±9.3	0.186
Nighttime PP	57.3±15.7	47.1±9.1	0.001	43.8±10.2	37.4±8.0	<0.001
% nocturnal decrease in SBP	4.8±10.0	9.3±7.8	0.004	4.1±10.5	8.3±8.4	0.012
% nocturnal decrease in DBP	9.2±10.4	13.6±9.3	0.014	10.1±9.6	14.2±9.3	0.019

Table 7. Office and 24-h ambulatory brachial and central BP in hypertensive patients based on the presence or absence of reduced renal function. Data are expressed as mean ± standard deviation. ABPM: ambulatory BP monitoring; DBP: diastolic blood pressure; PP: pulse pressure; SBP: systolic blood pressure

BP	Brachial			Central		
	With (n=28)	Without (n=180)	p value	With (n=28)	Without (n=180)	p value
Office SBP	141.6±22.9	136.1±17.9	0.148	131.2±27.2	125.0±15.6	0.260
Office DBP	85.2±12.7	87.3±11.3	0.360	86.9±12.5	88.6±11.4	0.493
Office PP	54.7±16.6	48.2±14.6	0.031	44.1±21.5	36.8±11.6	0.092
24-h SBP	131.3±12.1	129.0±14.0	0.409	121.3±16.1	118.8±12.3	0.362
24-h DBP	77.6±8.1	80.6±8.6	0.081	79.2±8.3	82.1±8.8	0.107
24-h PP	53.6±10.3	48.5±10.4	0.016	39.3±7.1	36.4±7.5	0.059
Daytime SBP	133.5±12.6	132.7±14.5	0.785	123.6±15.0	122.0±13.1	0.555
Daytime DBP	80.0±8.8	84.2±9.4	0.028	82.1±9.3	85.9±9.6	0.056
Daytime PP	54.0±10.4	48.5±10.6	0.012	38.7±7.9	35.8±7.8	0.066
Nighttime SBP	125.1±15.2	120.5±16.1	0.157	116.7±22.4	112.0±15.3	0.161
Nighttime DBP	71.7±9.4	72.5±9.4	0.676	72.0±9.2	73.6±9.7	0.403
Nighttime PP	53.4±11.5	48.1±11.0	0.020	41.8±10.1	37.9±8.4	0.031
% nocturnal decrease in SBP	6.1±8.7	9.0±8.3	0.090	5.8±10.2	8.0±9.0	0.226
% nocturnal decrease in DBP	10.1±9.5	13.5±9.8	0.087	12.0±8.9	13.9±9.7	0.345

BP was further analyzed based on the type of renal abnormality. Patients with either microalbuminuria or reduced renal function had higher brachial and central BP, as well as a blunted nocturnal BP decline, compared to their counterparts without such abnormalities. Differences were more pronounced when comparing patients with and without microalbuminuria (Table 6) than when comparing those with and without reduced renal function (Table 7). In this latter comparison, and as regards central BP, only nocturnal PP was significantly higher in patients with impaired eGFR.

Risk of the presence of renal organ damage based on office and 24-h ambulatory brachial and central BP

The association of renal organ damage (indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2) with office and 24-h ambulatory BP, both brachial and central, is shown in a Forest plot in Fig. 1. After adjusting for age, gender, and antihypertensive treatment, only nighttime brachial SBP and 24-h, daytime, and nighttime brachial PP were significantly associated with renal organ damage. As regards central BP indices, significant associations were again present for 24-h, daytime, and nighttime PP. Confidence intervals for odds ratios of brachial and central BP show overlap, indicating that there were no differences between brachial and central estimates. Moreover, as shown in Table 8, we calculated odds ratios for brachial and central BP estimates after simultaneous adjustments. None of the central estimates maintained its statistical significance after brachial adjustment.

Discussion

Classically, the diagnosis and management of hypertensive patients have been based on brachial or peripheral BP measurement in the office, and more recently this approach was extended to home measurement and 24-h ABPM. Technological advances have allowed the development of new devices capable of measuring central BP in a noninvasive way [13, 14]. Their development is based on the premise that central or aortic BP reflects the true load on the target organ. In fact, several studies have suggested that central BP is associated with the macrovascular damage, specifically cardiac and carotid damage, but is not so closely related to the microvascular lesion [2, 4,6, 15–18]. Kollias et al. analyzed four studies that showed similar correlations of urine albumin excretion with brachial SBP and central BP (three in

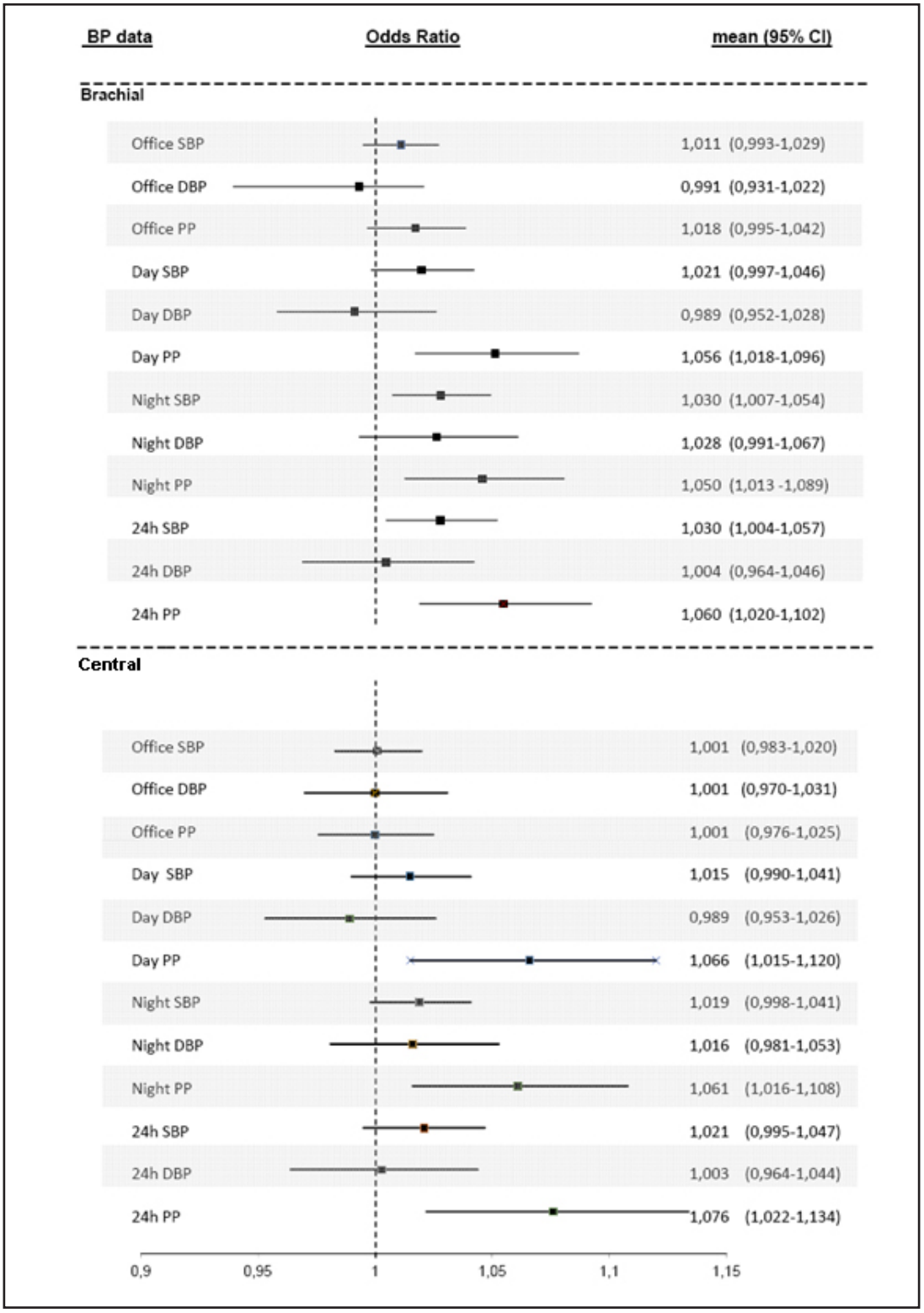

Fig. 1. Forest plot of the risk of renal organ damage (as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2) based on office and 24-h ambulatory brachial and central BP. DBP: diastolic blood pressure; PP: pulse pressure; SBP: systolic blood pressure.

Table 8. Odds ratio for each mmHg increase (95% confidence interval) of the association of each pair of blood pressure estimates (brachial and central), after adjustment for each other, with the presence of kidney damage. Data are expressed as: odds ratio (95% confidence interval). DBP: diastolic blood pressure; PP: pulse pressure; SBP: systolic blood pressure Odds ratio also adjusted for age, gender, and the use of antihypertensive treatment

	Office	24-hours	Day	Night
Brachial SBP	1.07 (1.00–1.13)	1.07 (1.00–1.14)	1.05 (0.99–1.11)	1.06 (1.01–1.12)
Central SBP	0.94 (0.88–1.01)	0.96 (0.90–1.03	0.97 (0.91–1.03)	0.97 (0.92–1.02)
Brachial DBP	0.91 (0.83–1.00)	1.04 (0.78–1.37)	1.02 (0.79–1.30)	1.14 (0.99–1.29)
Central DBP	1.10 (0.99–1.21)	0.97 (0.74–1.27)	0.98 (0.77–1.24)	0.91 (0.80–1.03)
Brachial PP	1.04 (1.00–1.09)	1.04 (0.92–1.15)	1.05 (0.96–1.15)	1.03 (0.98–1.09)
Central PP	0.97 (0.92–1.01)	1.03 (0.92–1.15)	1.01 (0.89–1.13)	1.03 (0.97–1.10)

an office setting and one with ABPM) [2]. Goupil et al. also found that microalbuminuria and early chronic kidney disease were not associated with office central BP [5]. A previous study from our group showed similar correlation of microalbuminuria with office central BP and office brachial BP [18]. Finally, in a Chinese community, Fan et al. found that, compared with office brachial SBP, office central SBP was a stronger predictor for kidney function decline [6]. In all the studies discussed, central BP was measured by radial tonometry.

The widespread use of 24-h ABPM has revealed the importance of the BP circadian rhythm and the role of nocturnal BP in the prognosis of organ damage in hypertension [19–21]. In the present study, we evaluated the possible relationship between renal organ damage and central ABPM hemodynamic indices in hypertensive patients.

Twenty-six percent of the hypertensive patients included in the study had kidney damage, as indicated by either microalbuminuria or eGFR <60 ml/min/1.73 m^2. These patients, as expected, had a worse CV profile, were older, had a higher BMI, and displayed higher rates of diabetes, dyslipidemia, and previous CV events.

Patients with renal damage showed worse BP control by ABPM, both brachial and central, mainly reflected in the SBP and PP values. It is known that DBP and mean BP are relatively constant along the vascular tree while the SBP increases as it moves away from the heart. Greater SBP amplification has been associated with vascular stiffness that induces higher PWV and early wave reflections [22]. Indeed, in this study patients with kidney damage showed an increase in aortic PWV, a known marker of vascular stiffness.

Regarding the BP circadian rhythm, it is known that patients with kidney damage show a blunted nocturnal decrease in brachial BP. In this study we confirmed this BP pattern, which was also altered at the central level [23, 24].

The main result of the study is that we did not detect significant differences between brachial and central BP indices in patients with renal damage. It is known that kidney damage in hypertension is related to the degree to which renal autoregulatory mechanisms fail to prevent the high BP from being transmitted to the renal microvasculature. Vasoconstrictions of the preglomerular vasculature protect the rest of the renal vasculature from elevated pressure transmission [25]. Therefore, the kidney may not represent a typical target organ directly exposed to aortic pressure. This work suggests that in hypertensive patients with renal damage, optimal control of 24-h brachial BP will be sufficient to ensure renal protection.

The study has some limitations: First, due to the cross-sectional design we could not establish causality on the level of central BP with the lesion or CV event (pathogenic factor) or regression of the target organ damage. Second, the number of patients with renal abnormalities (either microalbuminuria or reduced renal function) was relatively low. The study has, however, important strengths: (1) use of a calibrated device, (2) absence of observer influence on the BP result due to ease of use of the device, (3) simultaneous assessment of 24-h monitoring of both brachial and central BP, and (4) the fact that microalbuminuria and eGFR are reliable markers of kidney damage.

In summary, this study suggests that cuff-based oscillometric central ABPM does not

show superiority over brachial ABPM for the prediction of renal organ damage, as indicated by either microalbuminuria or eGFR, in treated hypertensive patients. More studies, however, need to be performed in a prospective and controlled manner in large hypertensive subpopulations to identify the real role of central BP in clinical practice, specifically in relation to BP control, target organ damage, CV events, CV risk evaluation, and guided pharmacological treatment.

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Disclosure Statement

None declared.

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