

ORIGINAL ARTICLE

Waiting a few extra minutes before measuring blood pressure has potentially important clinical and research ramifications

SB Nikolic¹, WP Abhayaratna², R Leano³, M Stowasser³ and JE Sharman¹

Office blood pressure (BP) is recommended to be measured after 5 min of seated rest, but it may decrease for 10 min of seated rest. This study aimed to determine the change (and its clinical relevance) in brachial and central BP from 5 to 10 min of seated rest. Office brachial and central BP (measured after 5 and 10 min), left ventricular (LV) mass index, 7-day home and ambulatory BP were measured in 250 participants with treated hypertension. Office brachial and central BP were significantly lower at 10-min compared with 5-min BP ($P < 0.001$). Seven-day home systolic BP (SBP) was significantly lower than office SBP measured at 5 min ($P < 0.001$), but was similar to office SBP at 10 min ($P = 0.511$). From 5 to 10 min, the percentage of participants with controlled BP increased and the percentage of participants with high central pulse pressure (PP) decreased ($P < 0.001$). Moreover, brachial and central PP were significantly correlated with LV mass index measured at 10 min ($r = 0.171$, $P = 0.006$ and $r = 0.139$, $P = 0.027$, respectively), but not at 5 min ($r = 0.115$, $P = 0.068$ and $r = 0.084$, $P = 0.185$, respectively). BP recorded after 10 min is more representative of true BP control. These findings have relevance to appropriate diagnosis of hypertension and design of clinical trials.

Journal of Human Hypertension (2014) 28, 56–61; doi:10.1038/jhh.2013.38; published online 30 May 2013

Keywords: blood pressure determination; pulse wave analysis; pulse pressure; left ventricular mass

INTRODUCTION

Current guidelines for the management of hypertension recommend that office brachial blood pressure (BP) should be optimally measured after 5 min of seated rest.^{1,2} However, brachial BP may decrease for up to 10 min of seated rest, after which it reaches a plateau level.^{3,4} Furthermore, office brachial BP averaged over 10 min of seated rest in the absence of an observer has been shown to closely relate to out-of-office BP (daytime ambulatory BP).⁵ This is an important finding because out-of-office BP measures (for example, 7-day home BP and daytime ambulatory BP) have been shown to be stronger predictors of cardiovascular risk when compared with office BP values.^{6,7} Consequently, office brachial BP measured after the recommended 5-min rest period may not be a good representative of true BP, and using this as the sole method to assess BP control may result in misclassification or inappropriate management of some individuals.⁸ The first aim of this study was to determine the change in seated brachial BP when measured after 5 and 10 min of seated rest. This study also sought to determine the clinical relevance of the change in brachial BP over time by assessing: (1) the change in BP classification; (2) the comparison with out-of-office BP; and (3) associations with BP-related end-organ damage (left ventricular (LV) mass).

Central BP may be significantly different from the brachial BP measured at the same time.⁹ However, central BP is pathophysiologically more important than brachial BP with respect to cardiovascular disease and has a stronger relation to LV mass than brachial BP.¹⁰ In recent years, large longitudinal studies have indicated that central BP has more power for predicting future cardiovascular events and mortality when compared with brachial BP.¹¹ Furthermore, central BP may also change differently from brachial BP in response to physiological

stimuli (for example, posture, medications and exercise).^{12,13} To our knowledge, there have been no studies examining how central BP acutely changes over 5 min compared with 10 min, and this was an additional aim of this study. As with the change in brachial BP over time, we also sought to determine the clinical relevance of central BP changes after 5 min compared with 10 min of seated rest.

MATERIALS AND METHODS

Study population

Data were analysed from baseline examination of 250 participants, aged 18–75 years, with treated essential hypertension who were enrolled in the BP GUIDE study ([http://www.anzctr.org.au; ACTRN12608000041358](http://www.anzctr.org.au;ACTRN12608000041358)). See Table 1 for participant characteristics. Exclusion criteria included taking more than three anti-hypertensive medications or office BP $> 180/100$ mm Hg, a history of coronary artery or renal disease, secondary hypertension, aortic valve stenosis or obstructive atherosclerosis at the upper limb (difference of > 20 mm Hg in systolic BP between both arms), severe LV hypertrophy (LV mass index, indexed to height^{2.7}, in women ≥ 59 g m^{-2.7} and men ≥ 64 g m^{-2.7} measured by echocardiography) and pregnant women. The study was approved by the local Human Research Ethics Committees of attending study sites. All participants gave informed consent and all the procedures were performed in accordance with the Declaration of Helsinki (2000).

Protocol

Study participants attended the research clinic at only one occasion in the morning where all measurements were acquired.

¹Menzies Research Institute Tasmania, University of Tasmania, Hobart, Tasmania, Australia; ²College of Medicine, Biology and Environment, Australian National University, Australian Capital Territory, Australia and ³Department of Medicine, University of Queensland, Brisbane, Queensland, Australia. Correspondence: Dr JE Sharman, Menzies Research Institute Tasmania, University of Tasmania, Private Bag 23, Hobart, Tasmania 7000, Australia.

E-mail: James.Sharman@menzies.utas.edu.au

Received 17 January 2013; revised 13 March 2013; accepted 26 March 2013; published online 30 May 2013

Table 1. Characteristics of study participants

Variable	Mean \pm s.d. or %
Age (years)	64 \pm 8
Gender (% male)	52
Body mass index (kg m ⁻²)	29.3 \pm 4.8
Waist-to-hip ratio	0.94 \pm 0.51
LV mass index (g m ^{-2.7})	32.7 \pm 8.9
Heart rate (b.p.m.)	68 \pm 10
Diabetes (%)	7.6
Glucose (mmol l ⁻¹)	5.9 \pm 6.1
Total cholesterol (mmol l ⁻¹)	5.13 \pm 1.02
Low-density lipoprotein (mmol l ⁻¹)	3.04 \pm 0.94
High-density lipoprotein (mmol l ⁻¹)	1.39 \pm 0.47
Triglycerides (mmol l ⁻¹)	1.65 \pm 1.11
Medications	
Angiotensin-converting enzyme inhibitors (%)	30
Angiotensin receptor inhibitors (%)	63
β -Blockers (%)	9
Statins (%)	30
Diuretics (%)	36

Participants were asked to avoid exercise on the day of examination, as well as caffeine-containing beverages, heavy meals and smoking at least 3 h before the visit. All BP measures were recorded by a trained (non-clinician) research technician in a quiet, temperature-controlled room. Brachial BP was recorded as the average of duplicate measures taken 1 min apart after 5 min of seated rest, as per recommendations. Duplicate measures of central BP were then recorded and calibrated to the averaged brachial BP obtained previously (approximately 5 min of seated rest). Tonometric measures took approximately 2–4 min. Following this, two measures of brachial BP recorded 1 min apart were acquired again, and the average values were used to recalibrate the central BP (approximately 10 min of seated rest). After acquiring the BP measures, a blood sample was collected for standard clinical biochemistry and two-dimensional echocardiography measures of LV mass were recorded. At the end of the clinic visit, each participant was fitted with a 24-h ambulatory BP device. They were also provided with a BP device for 7-day home BP monitoring together with instructions on how to measure this, commencing on the day after the clinical visit.

Office brachial BP

Upper arm BP was recorded using a validated semiautomated oscillometric device (Omron HEM-907; OMRON Europe BV (OMCE), Hoofddorp, The Netherlands).¹⁴ BP was measured in accordance with guidelines, using an appropriately sized cuff with the arm supported at the heart level.^{1,2} Participants were seated quietly in a chair with feet flat on the floor and back supported.^{1,2} Uncontrolled BP was defined as $\geq 140/90$ mm Hg as per accepted criteria.² Brachial pulse pressure (PP) was calculated as the difference between brachial systolic and diastolic BP.

Office central BP

Seated central BP was measured by radial applanation tonometry using validated and highly reproducible generalized transfer function (SphygmoCor 8.1; AtCor Medical, Sydney, Australia).^{15,16} Central BP was calibrated by two methods: (1) calibration with the brachial systolic and diastolic BP and (2) calibration with mean (1/3 PP + diastolic BP) and diastolic BP; and both were used for the analysis. Central PP was calculated as the difference between central systolic and diastolic BP. The Strong Heart Study¹⁷ showed that central PP ≥ 50 mm Hg independently predicted adverse cardiovascular outcomes. Accordingly, for the purpose of this study, we defined high central PP as ≥ 50 mm Hg.

Home BP

Seven-day home BP was self-measured using a validated oscillometric device (UA-767, A&D Mercury; A&D Medical, Thebarton, South Australia, Australia).¹⁸ Participants were instructed to take BP measures in a warm and quiet room after at least 5 min of seated rest in a chair enabling back support, with feet flat on the ground and the arm with the cuff supported at the heart level. Home BP was measured in duplicate in the morning (between 0600 and 1000 hours), mid-day and evening (between 1800 and 2200 hours). The first of two BP measurements was discarded and the second BP was recorded and used for analysis. Home BP was compared with office BP values to identify individuals with white coat hypertension (increased office and normal home BP) and masked hypertension (normal office and increased home BP). Raised home BP was defined as $\geq 135/85$ mm Hg.¹⁹

Twenty-four hour ambulatory BP

A validated device (TM-2430, A&D Mercury; A&D Medical)²⁰ was used for ambulatory BP monitoring. Participants were advised to maintain routine daily life activities and the device was set to record BP every 30 min during the day (0600–2200 hours) and every hour during the night (2200–0600 hours).²¹ Daytime BP was also compared with office BP values to identify individuals with white coat and masked hypertension. Raised daytime BP was defined as $\geq 135/85$ mm Hg.²¹

Echocardiography

LV mass was measured by two-dimensional echocardiography (Vivid 7; GE Medical Systems, Milwaukee, WI, USA), calculated by the method of Devereux and Reichek²² and indexed to height^{2,7} (LV mass index).

Blood biochemistry

A fasted or non-fasted blood sample was drawn to determine total, low-density and high-density lipoprotein cholesterol, glucose and triglycerides. Blood was analyzed as per standard hospital pathology laboratory procedures.

Statistical analysis

Statistical analyses were performed using SPSS for windows software version 17.0 (SPSS Inc., Chicago, IL, USA). Comparison of BP variables measured at 5 and 10 min were determined by independent *t*-tests. Between-group categorical variables were assessed by the χ^2 test. Correlations between continuous variables were assessed by Pearson's correlation. Difference in the strengths of associations between central and brachial PP with LV mass index were compared by calculation of Z-statistic scores. Multiple regression was used to identify predictors of the change in brachial systolic BP from 5 to 10 min seated rest. A value of $P < 0.05$ was taken as statistically significant.

RESULTS

BP changes over time

There were no significant differences in central BP values when calibrated using brachial systolic and diastolic BP compared with calibration using mean and diastolic BP. As shown in Table 2, both brachial and central BPs measured at 10 min were significantly lower than the BP values recorded at 5 min. Brachial PP was also significantly lower after 10 min compared with 5 min of seated rest. Central PP also declined over time, but this was of borderline significance.

Table 2. BP values measured at 5 and 10 min of seated rest

BP variable (mm Hg)	5 min	10 min	Change	P-value
Brachial SBP	131.3 ± 14.0	127.1 ± 13.5	-4.2 ± 6.3	<0.001
Brachial DBP	77.1 ± 9.7	75.4 ± 9.6	-1.8 ± 4.2	0.041
Brachial PP	54.2 ± 11.3	51.7 ± 11.2	-2.5 ± 5.8	0.015
Central SBP (calibration 1)	119.1 ± 13.7	115.5 ± 13.1	-3.7 ± 5.4	0.002
Central DBP (calibration 1)	78.1 ± 9.8	76.3 ± 9.7	-1.8 ± 4.1	0.035
Central PP (calibration 1)	41.0 ± 10.7	39.2 ± 10.4	-1.8 ± 4.4	0.054
Central SBP (calibration 2)	118.8 ± 13.1	115.2 ± 12.6	-3.6 ± 5.5	0.003
Central PP (calibration 2)	40.7 ± 10.7	38.9 ± 10.7	-1.7 ± 4.5	0.080

Abbreviations: BP, blood pressure; calibration 1, calibration by brachial systolic and diastolic BP; calibration 2, calibration by mean and diastolic BP; DBP, diastolic BP; PP, pulse pressure; SBP, systolic BP. Data are mean ± s.d.

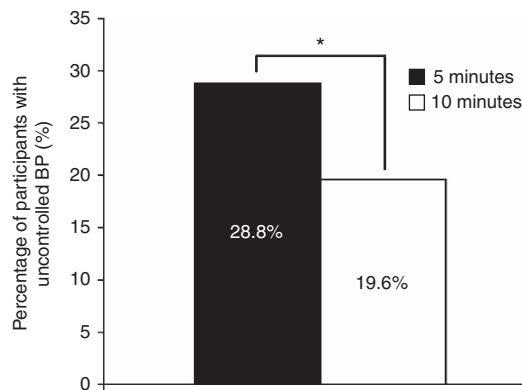


Figure 1. Percentage of study participants with uncontrolled BP when measured after 5 and 10 min of seated rest; * $P < 0.001$ and $n = 250$. From 5 to 10 min, there was a significant decrease in the percentage of individuals regarded as having uncontrolled BP.

Clinical relevance of the change in BP

Figure 1 shows the change in the distribution of participants with uncontrolled BP after 5 and 10 min of seated rest. There was a significant decrease over time in the number of participants regarded as having uncontrolled BP (from $n = 72$ to 49 ; $P < 0.001$). Also, when 7-day home BP was used to identify participants with white coat or masked hypertension, in the time period from 5 to 10 min of seated rest, there were 6% ($n = 15$) fewer participants classified as having white coat hypertension and 4% ($n = 10$) more participants classified as having masked hypertension (Figure 2). Furthermore, when we used daytime BP to identify these individuals, there were 4% ($n = 10$) less participants classified as having white coat hypertension and 8% ($n = 19$) more participants classified as having masked hypertension.

The change in the percentage of participants with high central PP after 5 and 10 min of seated rest is presented in Figure 3. There was a significant decrease of 6.4% ($n = 16$) in the number of participants with high central PP ($P < 0.001$) at 10-min compared with 5-min BP classification. When central PP calibrated with mean and diastolic BP was used, a significant decrease of 4.9% ($n = 13$) in the number of participants with high central PP ($P < 0.001$) was noticed.

Table 3 shows the correspondence between out-of-office BPs and the BPs recorded at different times. Brachial systolic BP values measured after 5- and 10-min rest were significantly lower than daytime ambulatory systolic BP. However, the brachial systolic BP at 10 min was similar to 7-day home systolic BP. On the other hand, brachial systolic BP measured at 5 min was significantly higher than 7-day home systolic BP. The average number of BP readings taken over the 24-h period for the study population was 41.7. From these, 1.5 readings (on average) were excluded because of measurement error, leaving 96.4% valid readings.

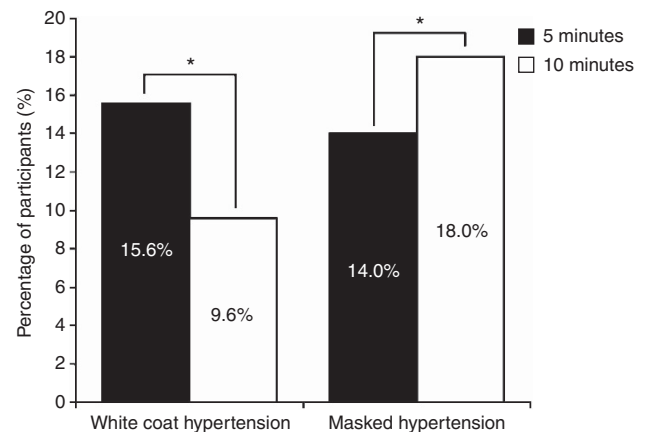


Figure 2. Percentage of study participants with white coat hypertension and masked hypertension when home BP was compared with 5- and 10-min brachial BP. From 5 to 10 min, there was a significant decrease (39 vs 24; $P < 0.001$) in the number of participants who were classified as having white coat hypertension and a significant increase (35 vs 45; $P < 0.001$) in the number of participants who were identified as having masked hypertension.

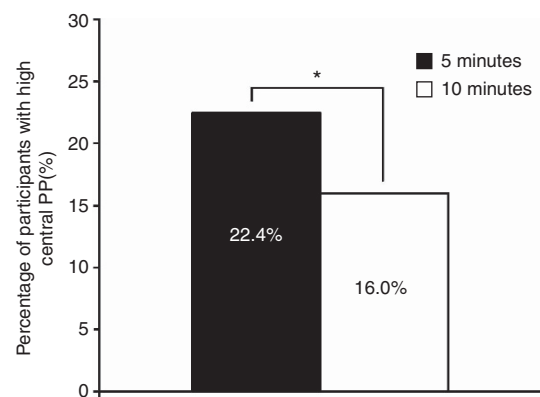


Figure 3. Percentage of study participants with high central PP when measured after 5 and 10 min of seated rest; * $P < 0.001$ and $n = 250$. Note a significant decrease in the percentage of participants with high central PP at 10 min compared with 5 min.

Table 4 shows the correlations between LV mass index and BP values. Brachial PP measured at 10-min seated rest was significantly correlated with LV mass index, whereas brachial PP measured at 5 min did not correlate with LV mass index. Similarly, 10-min central PP (calibrated by both methods) was significantly

Table 3. Daytime ambulatory and home SBP and their differences from office BP measured after 5 and 10 min of seated rest

BP variable	BP (mm Hg)	Mean difference from 5 min BP	P-value	Mean difference from 10 min BP	P-value
Daytime SBP	135.3 ± 11.7	- 3.9 ± 14.1	< 0.001	- 8.2 ± 13.5	< 0.001
Home SBP	127.7 ± 12.3	3.7 ± 14.6	< 0.001	- 0.6 ± 13.7	0.511

Abbreviations: BP, blood pressure; SBP, systolic blood pressure. Data are mean ± s.d.

correlated with LV mass index, whereas there was no significant correlation between 5-min central PP and LV mass index. The correlations between central systolic BP and PP with LV mass index (at both times) were slightly improved when calibration 2 was used and compared with calibration 1 method, but the difference in slopes were not of statistical significance ($P > 0.05$ for all). Similarly, the slope of relationships between central PP and brachial PP (at 10 min) with the LV mass index were not significantly different (calibration 1 central PP vs brachial PP, $Z = 0.520$, $P = 0.607$ and calibration 2 central PP vs brachial PP, $Z = 0.340$, $P = 0.734$). Neither brachial nor central systolic BPs were correlated with LV mass index at either time points, whereas brachial and central diastolic BPs were significantly correlated only when measured at 10 min ($r = -0.173$, $P = 0.006$ and $r = -0.171$, $P = 0.006$), similar to PP. On the other hand, LV mass index was significantly correlated with 7-day home systolic BP ($r = 0.140$, $P = 0.027$), 7-day home PP ($r = 0.145$, $P = 0.022$), mean 24-h systolic BP ($r = 0.175$, $P = 0.005$) and mean 24-h PP ($r = 0.259$, $P < 0.001$).

The change in brachial systolic BP from 5 to 10 min was significantly correlated with the use of angiotensin-converting enzyme inhibitors ($r = 0.212$, $P < 0.001$), brachial systolic BP at 5 min ($r = 0.302$, $P < 0.001$), brachial PP at 5 min ($r = 0.282$, $P < 0.001$), central systolic BP at 5 min ($r = 0.279$, $P < 0.001$) and central PP at 5 min ($r = 0.260$, $P < 0.001$). Owing to significant collinearity between BP variables, only the use of angiotensin-converting enzyme inhibitor and brachial systolic BP at 5 min were entered into a multiple regression model as independent predictors of the change in brachial systolic BP over time. The use of angiotensin-converting enzyme inhibitor ($\beta = 0.161$, $P = 0.008$) and brachial systolic BP at 5 min ($\beta = 0.273$, $P < 0.001$) were significant predictors of the change in brachial BP, but this was not a strong model (adjusted $R^2 = 0.109$, $P < 0.001$).

DISCUSSION

Office brachial BP is recommended to be measured after 5 min of seated rest.² The principal findings of this study were, firstly, that significant falls in office brachial and central BP were observed in the time period from 5 to 10 min of seated rest. Second, this drop in BP over time would have resulted in significant reclassification of BP control, and this was evident for both brachial and central BP values. Third, we observed similar values for out-of-office systolic BP and the brachial systolic BP recorded after 10 min, but not after the recommended 5 min of seated rest. Finally, evidence of end-organ damage was correlated with the BP (brachial and central) values recorded after 10 min, but not 5 min of seated rest. Thus, waiting a few minutes longer than the recommended 5 min before measuring BP appears to provide a better representation of true BP. These findings have relevance to appropriate diagnosis of hypertension as well as the design of clinical trials in which brachial and central BP are measured.

Office brachial BP changes over time

Numerous studies have reported a time-dependent decrease in seated brachial BP.^{3-5,23-26} The exact mechanisms of this BP drop are yet to be elucidated, but may be explained by a gradual decrease in systemic vascular resistance caused by hemodynamic

Table 4. The correlation coefficients of LV mass index with brachial and central BP values at 5 and 10 min of seated rest

BP variable	5 min		10 min	
	r-value	P-value	r-value	P-value
Brachial SBP	0.012	0.851	0.020	0.752
Brachial DBP	- 0.118	0.061	- 0.172	0.006
Brachial PP	0.115	0.068	0.171	0.006
Central SBP (calibration 1)	0.017	0.789	0.016	0.803
Central PP (calibration 1)	0.084	0.185	0.139	0.027
Central SBP (calibration 2)	0.011	0.862	0.012	0.876
Central PP (calibration 2)	0.120	0.057	0.165	0.009

Abbreviations: calibration 1, calibration by systolic and diastolic BP; calibration 2, calibration by mean and diastolic BP; DBP, diastolic BP; PP, pulse pressure; SBP, systolic blood pressure.

modifications to the seated position.³ Relaxation of the patient and sympathetic withdrawal over time may also be a potential cause of BP reduction.³ The magnitude of the brachial BP change over time has been consistently described in the literature. A large population study in 5999 participants reported a significant decrease (10.3 mmHg in men and 10.4 mmHg in women) in brachial systolic BP (but no change in diastolic BP) over 25 min of seated rest.²³ This change resulted in a decrease in the prevalence of isolated systolic hypertension. Sala *et al.*³ reported a similar decrease in systolic BP (10.7 ± 1.0 mmHg) and also a decrease in diastolic BP (3.4 ± 0.6 mmHg) in the period from 2 to 16 min of seated rest. These investigators also estimated that the bulk of the fall in systolic BP (75.3%) and diastolic BP (71.7%) occurred in the initial 10 min of seated rest. This finding has been repeated in a recent study by van der Wel *et al.*,⁴ who also showed that the plateau level of the systolic BP decline occurred after about 10 min of seated rest. Our findings are in agreement with these previous studies, but have also focused on the potential clinical impact of the change in BP from 5 to 10 min of seated rest. Taken together, these studies suggest that 5 min is not a long enough period to wait before office BP measurements are acquired.

Clinical implications of BP changes over time

The change in brachial BP over time has potential clinical relevance as it may affect the evaluation and diagnosis of hypertension. Our study showed that if only the BP after 5-min rest was considered, more participants would be classified as having uncontrolled BP compared with the BP at 10 min. Indeed, 9.2% ($n = 23$) were reclassified from uncontrolled office BP to controlled office BP when the BP at 10 min was taken into account. In the clinical environment, this differential diagnosis could conceivably result in different (and possibly inappropriate) management of some individuals. A possible reason contributing to this change in classification could be related to a 'white coat effect'²⁷ given that this was 6% more likely if only the BP at 5 min was considered. Our finding on the reclassification regarding prevalence of masked hypertension is also of potential clinical relevance.²⁸ An argument may be raised that it would be better to overestimate BP in these patients, as this may increase the chances for more appropriate diagnosis and treatment. Even

though only a small number of participants were reclassified to having masked hypertension according to the 10-min BP level (4–8% depending on whether home or 24-h ambulatory BP was used to determine out-of-office BP), this finding provides additional support for routinely undertaking out-of-office BP to diagnose hypertension,⁷ which may be particularly relevant in patients with type 2 diabetes.²⁹

Out-of-office BP recordings (home and daytime ambulatory BP) have been shown to be stronger predictors of cardiovascular risk than office brachial BP.^{6,7} The lower prognostic value of office BP compared with out-of-office BP readings could potentially be attributed to inadequate time allowed before taking office BP recordings. Importantly, Myers *et al.*⁵ have showed that office brachial BP, recorded by an automated device and averaged over 10 min with the patient resting alone in a clinic room, was more similar with ambulatory daytime BP compared with standard 5-min BP measurements taken by either physician or technician.⁵ Although the 10-min automated office BP was more similar to ambulatory daytime BP, the automated BP was significantly lower even after correction for multiple comparisons.⁵ While the additional amount of time spent waiting may have contributed to the lower automated office BP readings at 10 min, the absence of the observer in itself may independently contribute to lower automated office BP readings. Our study is in agreement with the study of Myers *et al.*,⁵ as we have also found that our 10-min office BP was lower than daytime ambulatory BP. Although the difference between these two BP measurements is greater in our study, this could be explained by different settings in which BP measurements were taken (patient's physician referred ambulatory BP unit vs research setting) and different study population (hypertensive participants with nearly half untreated vs all treated hypertensive participants) and this may have contributed to our lower office BP values. Furthermore, we have also showed that brachial BP measured at 10 min (but not 5 min) were more similar to the average of 7-day resting home BP readings than BP measurements taken during daytime ambulatory condition. Thus, waiting for extra time, taking more readings and bringing the office BP closer to out-of-office BP values may potentially improve the utility of office BP measurements.⁵

Central BP indices have been shown to correlate independently with severe cardiovascular events and mortality.^{10,11} While yet to be incorporated into general medicine, the consideration of central BP may improve identification and management of patients with hypertension or increased cardiovascular risk.^{9,11,30,31} Indeed, two individuals with similar brachial systolic BP may have significantly different central systolic BP and, accordingly, may be classified into different risk groups related to BP.⁹ Furthermore, different BP-lowering drugs can differentially affect central BP compared with brachial BP and, looking to the future, this should be taken into account when assessing and deciding appropriate treatment options.^{12,32,33} In our study, we have shown for the first time that central BP decreased over time, similarly to brachial BP. Our data indicate the potential for overestimating risk related to BP when based on 5-min central PP readings, as 6.4% less individuals would have been identified with high central PP on the basis of the 10-min readings.¹⁷

Limitations

Our study represents a cross-sectional analysis of data from patients with treated hypertension, and the findings may not be generalizable to other populations. Different oscillometric devices were used to measure office and home BP. Although both devices have been validated,^{14,18} the use of separate machines might have affected study results. Central BP was measured by radial applanation tonometry using a generalized transfer function. Although this method has been validated and shown to be highly reproducible,^{15,16} calibration of the waveform using brachial

BP may have resulted in error of central BP estimation.³⁴ Furthermore, radial pressure waveforms were acquired only at one time point (after the BP measurements at 5 min) and the BP recorded at approximately 10 min was used to recalibrate the radial pressure waveforms. Since another tonometry reading was not acquired after 10 min, we may expect an underestimation of the change in central BP from 5 to 10 min of seated rest, but this needs to be tested in another study. In contrast to some previous studies,³⁵ the home BP values in our study were lower than ambulatory BP measurements. This might be explained by the protocol used for home BP measurements, whereby the first BP reading was discarded from each duplicate reading. This is not entirely in keeping with European guidelines;¹⁹ however, the reason for this approach was based on the evidence that the first reading (morning and evening) has been shown to be persistently higher than any subsequent BP measurement, and the average of later measurements is a stronger correlate of ambulatory BP.²⁴

In conclusion, our study showed that both brachial and central BP decreased significantly when measured a few minutes after the recommended 5 min of seated rest in individuals treated for hypertension. This change in BP over time may have significant implications when assessing the BP-related risk and managing patients with hypertension. Given the increasing body of evidence indicating the superiority of out-of-office BP values in predicting cardiovascular risk and total mortality,^{6,7} any advances that enable measurement of an office BP value that is closer to out-of-office BPs would be a valuable advancement in the clinical assessment of BP control. Although studies will be required to confirm this, our finding that 10-min office BP was similar to 7-day home BP measurements may suggest that office BP measured after 10 min may provide better prognostic information about cardiovascular risk than the conventional 5 min wait period before measuring BP. In any case, this study tends to support the work by Myers *et al.*⁵ that multiple, automated office BP measurements with the patient sitting alone in a quiet examining room could be the most appropriate method to measure office BP.

What is known about the topic

- Office brachial BP is recommended to be measured after 5 min of seated rest, but BP may continue to decrease for 10 min.
- The change in brachial BP over time has potential clinical relevance as it may affect the evaluation and diagnosis of hypertension.
- Central BP is a stronger predictor of mortality than brachial BP. To our knowledge, changes in office central BP over time and the clinical relevance of this change have never been assessed before.

What this study adds

- BP recorded after 10 min (but not 5 min) is more representative of true BP control and better correlates with end-organ damage.
 - Our findings have relevance to appropriate diagnosis of hypertension as well as the design of clinical trials in which brachial and central BP are measured.
-

CONFLICT OF INTEREST

Dr Sharman has research collaborations with AtCor Medical. The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

This study was supported, in part, by a National Health and Medical Research Council project grant (reference 569669), Canberra, Australia. Dr Sharman was supported by a National Health and Medical Research Council Career Development Award (reference 569519).

REFERENCES

- Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN *et al*. Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation* 2005; **111**(5): 697–716.
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL *et al*. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; **289**(19): 2560–2572.
- Sala C, Santin E, Rescaldani M, Magrini F. How long shall the patient rest before clinic blood pressure measurement? *Am J Hypertens* 2006; **19**(7): 713–717.
- van der Wel MC, Buunk IE, van Weel C, Thien TA, Bakx JC. A novel approach to office blood pressure measurement: 30-minute office blood pressure vs daytime ambulatory blood pressure. *Ann Fam Med* 2011; **9**(2): 128–135.
- Myers MG, Valdivieso M, Kiss A. Use of automated office blood pressure measurement to reduce the white coat response. *J Hypertens* 2009; **27**(2): 280–286.
- Niiranen TJ, Hanninen MR, Johansson J, Reunanen A, Jula AM. Home-measured blood pressure is a stronger predictor of cardiovascular risk than office blood pressure: the Finn-Home study. *Hypertension* 2010; **55**(6): 1346–1351.
- Hansen TW, Kikuya M, Thijs L, Bjorklund-Bodegard K, Kuznetsova T, Ohkubo T *et al*. Prognostic superiority of daytime ambulatory over conventional blood pressure in four populations: a meta-analysis of 7030 individuals. *J Hypertens* 2007; **25**(8): 1554–1564.
- Verberk WJ, Kroon AA, Lenders JW, Kessels AG, van Montfrans GA, Smit AJ *et al*. Self-measurement of blood pressure at home reduces the need for antihypertensive drugs: a randomized, controlled trial. *Hypertension* 2007; **50**(6): 1019–1025.
- Sharman J, Stowasser M, Fassett R, Marwick T, Franklin S. Central blood pressure measurement may improve risk stratification. *J Hum Hypertens* 2008; **22**(12): 838–844.
- Sharman JE, Fang ZY, Haluska B, Stowasser M, Prins JB, Marwick TH. Left ventricular mass in patients with type 2 diabetes is independently associated with central but not peripheral pulse pressure. *Diabetes Care* 2005; **28**(4): 937–939.
- Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. *Eur Heart J* 2010; **31**(15): 1865–1871.
- Williams B, Lacy PS, Thom SM, Cruickshank K, Stanton A, Collier D *et al*. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. *Circulation* 2006; **113**(9): 1213–1225.
- Sharman JE, McEniery CM, Campbell RI, Coombes JS, Wilkinson IB, Cockcroft JR. The effect of exercise on large artery haemodynamics in healthy young men. *Eur J Clin Invest* 2005; **35**(12): 738–744.
- El Assaad MA, Topouchian JA, Darne BM, Asmar RG. Validation of the Omron HEM-907 device for blood pressure measurement. *Blood Pressure Monit* 2002; **7**(4): 237–241.
- Holland DJ, Sacre JW, McFarlane SJ, Coombes JS, Sharman JE. Pulse wave analysis is a reproducible technique for measuring central blood pressure during hemodynamic perturbations induced by exercise. *Am J Hypertens* 2008; **21**(10): 1100–1106.
- Sharman JE, Lim R, Qasem AM, Coombes JS, Burgess MI, Franco J *et al*. Validation of a generalized transfer function to noninvasively derive central blood pressure during exercise. *Hypertension* 2006; **47**(6): 1203–1208.
- Roman MJ, Devereux RB, Kizer JR, Okin PM, Lee ET, Wang W *et al*. High central pulse pressure is independently associated with adverse cardiovascular outcome the strong heart study. *J Am Coll Cardiol* 2009; **54**(18): 1730–1734.
- Rogoza AN, Pavlova TS, Sergeeva MV. Validation of A&D UA-767 device for the self-measurement of blood pressure. *Blood Press Monitor* 2000; **5**(4): 227–231.
- Parati G, Stergiou GS, Asmar R, Bilò G, de Leeuw P, Imai Y *et al*. European Society of Hypertension guidelines for blood pressure monitoring at home: a summary report of the Second International Consensus Conference on Home Blood Pressure Monitoring. *J Hypertens* 2008; **26**(8): 1505–1526.
- Palatini P, Frigo G, Bertolo O, Roman E, Da Corta R, Winnicki M. Validation of the A&D TM-2430 device for ambulatory blood pressure monitoring and evaluation of performance according to subjects' characteristics. *Blood Press Monitor* 1998; **3**(4): 255–260.
- Head GA, McGrath BP, Mihailidou AS, Nelson MR, Schlaich MP, Stowasser M *et al*. Ambulatory blood pressure monitoring in Australia: 2011 consensus position statement. *J Hypertens* 2012; **30**(2): 253–266.
- Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation* 1977; **55**(4): 613–618.
- van Loo JM, Peer PG, Thien TA. Twenty-five minutes between blood pressure readings: the influence on prevalence rates of isolated systolic hypertension. *J Hypertens* 1986; **4**(5): 631–635.
- Verberk WJ, Kroon AA, Kessels AG, Lenders JW, Thien T, van Montfrans GA *et al*. The optimal scheme of self blood pressure measurement as determined from ambulatory blood pressure recordings. *J Hypertens* 2006; **24**(8): 1541–1548.
- Myers MG, Godwin M, Dawes M, Kiss A, Tobe SW, Grant FC *et al*. Conventional versus automated measurement of blood pressure in primary care patients with systolic hypertension: randomised parallel design controlled trial. *BMJ* 2011; **342**: d286.
- Mancia G, Bertinieri G, Grassi G, Parati G, Pomidossi G, Ferrari A *et al*. Effects of blood-pressure measurement by the doctor on patient's blood pressure and heart rate. *Lancet* 1983; **2**(8352): 695–698.
- Pickering TG, Gerin W, Schwartz JE, Spruill TM, Davidson KW, Franz Volhard lecture: should doctors still measure blood pressure? The missing patients with masked hypertension. *J Hypertens* 2008; **26**(12): 2259–2267.
- Ohkubo T, Kikuya M, Metoki H, Asayama K, Obara T, Hashimoto J *et al*. Prognosis of 'masked' hypertension and 'white-coat' hypertension detected by 24-h ambulatory blood pressure monitoring 10-year follow-up from the Ohasama study. *J Am Coll Cardiol* 2005; **46**(3): 508–515.
- Franklin SS, Thijs L, Li Y, Hansen TW, Boggia J, Liu Y *et al*. Masked hypertension in diabetes mellitus: treatment implications for clinical practice. *Hypertension* 2013; **61**(5): 964–971.
- Wang KL, Cheng HM, Chuang SY, Spurgeon HA, Ting CT, Lakatta EG *et al*. Central or peripheral systolic or pulse pressure: which best relates to target organs and future mortality? *J Hypertens* 2009; **27**(3): 461–467.
- Schultz MG, Gilroy D, Wright L, Bishop WL, Abhayaratna WP, Stowasser M *et al*. Out-of-office and central blood pressure for risk stratification: a cross-sectional study in patients treated for hypertension. *Eur J Clin Invest* 2012; **42**(4): 393–401.
- Hirata K, Vlachopoulos C, Adji A, O'Rourke MF. Benefits from angiotensin-converting enzyme inhibitor 'beyond blood pressure lowering': beyond blood pressure or beyond the brachial artery? *J Hypertens* 2005; **23**(3): 551–556.
- Morgan T, Lauri J, Bertram D, Anderson A. Effect of different antihypertensive drug classes on central aortic pressure. *Am J Hypertens* 2004; **17**(2): 118–123.
- Hope SA, Meredith IT, Cameron JD. Effect of non-invasive calibration of radial waveforms on error in transfer-function-derived central aortic waveform characteristics. *Clin Sci (London, England: 1979)* 2004; **107**(2): 205–211.
- Saladini F, Benetti E, Malipiero G, Casiglia E, Palatini P. Does home blood pressure allow for a better assessment of the white-coat effect than ambulatory blood pressure? *J Hypertens* 2012; **30**(11): 2118–2124.