Research Report

Journal of International Medical Research
2017, Vol. 45(1) 123–133

DOI: 10.1177/0300060516677175
journals.sagepub.com/home/imr

Association of blood pressure in the supine position with target organ damage in subjects over 60 years old

Feng Wang[1], Hualing Zhao[2], Chao Yang[3], Guilan Kong[4], Lu Song[2], Chunhui Li[2], Yiming Wang[2], Shuohua Chen[5], Jing Wang[4] and Shouling Wu[5]

Abstract

Objective: To explore the correlation between blood pressure in the supine position and target organ damage in subjects over 60 years of age.

Methods: In 2444 individuals, we investigated the association of systolic blood pressure (SBP) in the supine position with the target organ damage indices microalbuminuria (ALBU), brachial–ankle pulse wave velocity (baPWV), and carotid intima–media thickness (IMT). Supine hypertension (SH) is defined as systolic blood pressure $\geq$ 140 mmHg and/or diastolic blood pressure $\geq$ 90 mmHg. Subjects were assigned to either the SH group (1275 cases) or the non-SH group (1169 cases).

Results: The levels of ALBU, baPWV, and IMT, as well as the percentage of participants with ALBU > 30 mg/L, baPWV $\geq$ 1400 cm/s, and IMT $\geq$ 1 mm, were significantly higher in the SH group than in the non-SH group. Multivariate logistic regression analysis showed that SH was an independent risk factor for baPWV and IMT, but the relationship with ALBU was not statistically significant after correction for confounding factors.

Conclusions: SH is a risk factor for target organ damage, as expressed by the indices baPWV and IMT. The association of SH with kidney damage requires further study.

[1]Sixth Hospital of Peking University, Beijing, China
[2]North China University of Science and Technology, Tangshan, China
[3]Renal Division, Department of Medicine, Peking University First Hospital, Peking University Institute of Nephrology, Beijing, China
[4]Medical Informatics Center, Peking University, Beijing, China
[5]Cardiology Department of Hebei United University, Affiliated Kailuan General Hospital, Tangshan, China

Registration number: ChiCTR-TNC-11001489
Registration date: 2011/08/30
Registration URL: http://www.chictr.org.cn/showproj.aspx?proj=8050

Corresponding authors:
Jing Wang, Medical Informatics Center, Peking University, Beijing 100191, China; Cardiology Department of Hebei United University, Affiliated Kailuan General Hospital, Tangshan 063000, China.
Emails: bjmu-wangjing@163.com; drwusl@163.com

Keywords
Aged, arteriosclerosis, hypertension, supine position, renal insufficiency, chronic

Date received: 1 June 2016; accepted: 11 October 2016

Introduction

Blood pressure can be categorized as sitting, supine, or orthostatic according to the position of measurement. Notable differences have been observed between sitting and supine blood pressures,[1] and human bodies spend one-third of the time in the supine position. Clinical practice and related research often use sitting blood pressure to diagnose hypertension, whereas less attention has been paid to supine blood pressure or supine hypertension (SH). SH is defined as systolic blood pressure (SBP) $\geq$ 140 mmHg and/or diastolic blood pressure (DBP) $\geq$ 90 mmHg.[2] The incidence of SH in patients with autonomic failure is approximately 50%.[3]

Previous studies have demonstrated that hypertension is a risk factor for cardiovascular dysfunction and is closely related to the onset of chronic kidney disease,[4] atherosclerosis,[5] arteriosclerosis,[6] and other target organ damage. However, the aforementioned studies were mostly based on sitting clinic[7,8] or home blood pressure,[9,10] or 24-h ambulatory monitoring.[10–12] Although studies have reported that SH is closely related to kidney damage (glomerular filtration rate)[13,14] and left ventricular hypertrophy,[15] research was based on relatively small sample populations. Very few large-scale studies have analysed both SH and target-organ damage indices, such as microalbuminuria (ALBU), brachial–ankle pulse wave velocity (baPWV), and carotid intima–media thickness (IMT). Therefore, this study evaluated data collected from Kailuan General Hospital (registration number: chiCTR-TNC-1100 1489) and examined the association of SH and indices of target-organ damage (ALBU, baPWW, and IMT) in subjects over 60 years of age.

Materials and methods

Study population

From 2006 to 2007, data were collected from the health examinations of current and retired employees of Kailuan Group at 11 hospitals, including Kailuan General Hospital, Kailuan Linxi Hospital, Kailuan Zhaogezhuang Hospital, Kailuan Tangjiazhuang Hospital, Kailuan Fangezhuang Hospital, Kailuan Lujiatuo Hospital, Kailuan Jinggezhuang Hospital, Kailuan Linnancang Hospital, Kailuan Qianjiaing Hospital, Kailuan Majiagou Hospital, and Kailuan rehabilitation hospital. The same group of employees underwent two more examinations in 2008–2009 and 2010–2011, conducted by the same medical staff as at the first examination, at the same locations, and with the same time sequence, questionnaire, and anthropometric and biochemical analysis methods as in the first health examination.

During the third health examination, retired employees of Kailuan Group ($\geq$ 60 years old) who underwent their examinations at Kailuan General Hospital, Kailuan Linxi Hospital, or Kailuan Zhaogezhuang Hospital were selected as study candidates by cluster sampling, with 25% of the population being selected at random. The study subjects underwent physical examinations and, with their consent, rescheduled a new appointment (the third + health examination) for blood pressure measurement in different positions, ultrasound examination of cervical blood vessels, 24-h ambulatory blood pressure monitoring, pulse wave velocity examination, and urinary microprotein detection. The study was approved by the ethics committees of Kailuan Group Hospital.

Inclusion and exclusion criteria

Inclusion criteria: 1. retired workers ≥ 60 years of age who underwent their health examinations at the Kailuan General Hospital, Kailuan Linxi Hospital, or Kailuan Zhaogezhuang Hospital; 2. subjects without severe disabilities who could stand and walk in examinations by themselves; 3. subjects without defects in cognitive ability who could complete the questionnaire; and 4. subjects who agreed to participate in the study and provided signed informed consent.

Exclusion criteria: 1. valvular heart disease or cardiomyopathy; 2. frequent premature beats (all types of beats > 6 times/min); 3. atrial fibrillation, or atrioventricular or intraventricular conduction block; and 4. exposure to antipsychotic drugs, anti-Parkinson drugs, antidepressants, or sedative drugs within the previous 2 weeks.

Data collection

The content of the epidemiological survey, anthropometric indicators, and biochemical testing methods used in the current study can be found in our previous study.[16] Smoking was defined as at least one cigarette per day on average for at least 1 year; drinking was defined as drinking at least 100 mL of liquor (>50% alcohol content) per day on average for at least 1 year; and exercise was defined as aerobic activities (e.g., walking, jogging, ball games, swimming) at least three times a week for at least 30 minutes.

Sitting and supine blood pressure measurements

During the third + health examination, blood pressure was measured after subjects had been sitting calmly for 15 min and had refrained from smoking or consuming tea or coffee for 30 min before measurement. A calibrated mercury sphygmomanometer (Yuwell, Yuyue, Jiangsu, China) was used to measure blood pressure in the right brachial artery, with systolic SBP readings taken at the first and DBP readings at the fifth Korotkoff sound. Blood pressure was measured three consecutive times at intervals of 1–2 min, and the average value was recorded. Subjects were then asked to adopt the supine position, and the right brachial pressure was measured by trained and certified observers after 5 min. Blood pressure measurements were obtained according to a standard protocol recommended by the American Heart Association.[29] The diagnostic criteria for sitting hypertension were SBP ≥ 140 mmHg and/or DBP≥ 90 mmHg, a history of hypertension, or a diagnosis of hypertension and the taking of related medication. The diagnostic criteria for SH were SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg.

Laboratory testing

On the morning of the third examination, after fasting for 8 h, 5 mL of venous blood was collected from each subject into a vacuum tube and centrifuged at 3000 g for 10 min at room temperature (24°C), with upper serum collected for measurement within 4 h. Clinical laboratories measured serum creatinine levels by the colourimetric method using a liquid double dosage form creatinine assay kit manufactured by Beikong Biotechnology Co., Ltd (production batch number: 150203; batch variation coefficient < 5%; Changping, China). Analysis of other biochemical parameters, including fasting blood glucose (FBG), low-density lipoprotein cholesterol (LDL-C), and high-sensitivity C-reactive protein (hsCRP), was conducted on a Hitachi automatic analyser (7600 Automatic Analyzer; Hitachi, Tokyo, Japan). From the third + physical examination onwards, ALBU was measured using the Uppergold U2 quantitative reader manufactured by (Shanghai Upper Bio-Tech Pharmaceutical

Co. Ltd., Shanghai, China). In this study, increased ALBU was defined as ALBU $\geq$ 30 mg/L.[17]

Brachial–ankle pulse wave velocity

From the third + physical examination onwards, baPWV was measured using a BP-203RPE3 noninvasive vascular screening device (Omron Healthcare Co. Ltd, Tokyo, Japan). The data were read directly via network connections. The examination room temperature was maintained in the range 22–25 °C. The participant was requested not to smoke prior to the examination and had rested for at least 5 min. The pillow was removed to ensure a supine posture, with palms facing up and placed at the sides of the body. Sphygmomanometer cuffs were wrapped around the upper arms and the lower ankles. The label of the upper arm cuff was aligned with the brachial arteries, with the lower edge of the cuff 2–3 cm from the cubital fossa strips. The label of the lower limb cuff was positioned on the medial side of the leg, with the lower edge of the cuff 1–2 cm from the malleolus. A phonocardiography transducer was placed over the precordium. Electrocardiogram (ECG) electrodes were clipped to the left and the right wrists. Two measurements were taken per participant, and the second measurement was used as the final result. This study used the greater of the baPWV values of the left and right sides for further analyses.[18]

Carotid artery intima–media thickness

From the third + physical examination onwards, test participants were examined by physicians with more than 5 years of clinical experience in ultrasonography and who had undergone the standard training. A Philips HD-15 colour ultrasound diagnostic apparatus (Koninklijke Philips Electronics, Amsterdam, Netherlands) with a 5–12 MHz high-frequency probe was used for the measurement. The test participant assumed the supine position with the head turned toward the contralateral side from the examination. In the 1–2 cm segment at the end of the left and the right common carotid artery, IMT was measured in the longitudinal section of the rear wall (to avoid plaque). One person operated the apparatus and the other recorded the results. After the two physicians had confirmed their observations, detailed results were recorded. In accordance with the method of Hedblad et al.,[19] the larger of the values for the left and the right common carotid artery was used for analyses. A common carotid IMT $\geq$ 1.00 mm was defined as an increase in IMT.

Statistical analyses

The annual physical examination data from the years 2006–2007, 2008–2009, and 2010–2011 were obtained from the hospitals that performed the examinations. These data were uploaded via network to the Kailuan General Hospital server room and stored in an Oracle 10.2 g database. SPSS 13.0 statistical software (SPSS Inc., Chicago, IL, USA) was used for the statistical analyses. Normally distributed data were expressed as mean $\pm$ standard deviation. The *t*-test was used to compare data between two groups. Measurement data that formed a skewed distribution were analysed by *t*-test after logarithmic conversion. Count data were presented as n (%) and differences between groups were evaluated using the χ^2 test. Rank-correlation analysis was used to test for correlations between SBP and ALBU, baPWV, and IMT. Multivariate logistic regression was used to analyse the influence of SH on ALBU, baPWV, and IMT. $P < 0.05$ (bilateral) was considered statistically significant throughout.

Results

A total of 12,257 individuals (aged $\geq$ 60 years, and retired), accounting for 52.67%

of total retirees aged ≥ 60 years who had three physical examinations in Kailuan Group, underwent physical examinations at the three aforementioned hospitals. Of these, 3064 (25% of the total) were screened and 2860 subjects agreed to participate in this research (response rate: 93.34%). However, 46 volunteers dropped out for a variety of reasons, leaving 2814 (actual response rate: 91.84%), of whom 350 met the exclusion criteria and were excluded. Thus, the actual cohort comprised 2464 participants. Of these, 13 participants lacked SBP data and 7 had no DBP data, leaving a total of 2444 participants with complete blood pressure data.

General characteristics

The 2444 participants had a mean age of 67.40 years and included 1656 males. The age, supine/sitting SBP and DBP, supine heart rate, body mass index (BMI), FBG, log-converted (Lg) triglyceride (TG), Lg hsCRP, LDL-C, number of males, alcohol consumption, and percentage of participants taking antihypertensive medications were significantly higher in the SH group than in the non-SH group ($P < 0.05$). The differences in the proportion of smokers and exercisers between the SH group and the non-SH group were not statistically significant (Table 1).

Target organ damage indices

For the 2444 participants, the average levels of ALBU, baPWV, and IMT were 28.30 mg/L, 1795.95 cm/s, and 0.92 mm, respectively. The levels of ALBU, baPWV, and IMT, as well as the percentage of participants with ALBU > 30 mg/L,

Table 1. General participant data.

Variable	Total population ($n = 2444$)	Non-SH ($n = 1169$)	SH ($n = 1275$)	*P*
Males (%)	1656 (67.8)	755 (64.6)	901 (70.7)	<0.01
Age (years)	67.40 ± 6.04	66.72 ± 5.97	68.03 ± 6.04	<0.01
Sitting SBP (mmHg)	143.49 ± 20.93	129.15 ± 13.79	156.63 ± 17.44	<0.01
Sitting DBP (mmHg)	84.52 ± 11.27	78.95 ± 8.43	89.64 ± 11.13	<0.01
Supine SBP (mmHg)	138.95 ± 20.31	122.83 ± 10.51	153.73 ± 15.26	<0.01
Supine DBP (mmHg)	80.41 ± 10.88	74.25 ± 7.45	86.05 ± 10.48	<0.01
Supine heart rate (beats/minute)	74.14 ± 11.56	72.57 ± 10.92	75.58 ± 11.94	<0.01
BMI (kg/m^2)	25.35 ± 3.39	24.72 ± 3.18	25.93 ± 3.47	<0.01
FBG (mL/L)	5.89 ± 1.66	5.74 ± 1.44	6.03 ± 1.83	<0.01
Lg TG	0.30 ± 0.54	0.26 ± 0.54	0.34 ± 0.54	<0.01
Lg hsCRP	0.29 ± 1.05	0.24 ± 1.06	0.34 ± 1.03	0.04
LDL-C (mmol/L)	2.28 ± 0.84	2.79 ± 0.74	2.89 ± 0.91	0.01
Smokers (%)	536 (21.9)	256 (21.9)	280 (22.0)	0.97
Drinkers (%)	454 (18.6)	198 (16.9)	256 (20.1)	0.04
Exercising (%)	1388 (56.8)	664 (56.8)	724 (56.8)	0.99
Antihypertensive medications (%)	544 (22.3)	169 (14.5)	375 (29.4)	<0.01

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; Lg hsCRP, log-converted hypersensitive C-reactive protein; LgTG, log-converted triglycerides; SBP, systolic blood pressure; SH, supine hypertension. 1 mmHg = 0.133 kPa.

baPWV ≥ 1400 cm/s, and IMT ≥ 1 mm, were significantly higher in the SH group than in the non-SH group ($P < 0.01$) (Table 2).

Analysis of factors associated with different target organ damage indices

The participants' supine/sitting SBP and supine heart rate were positively correlated

Table 2. Target organ damage indices of participants.

Variable	Total population ($n = 2444$)	Non-SH ($n = 1169$)	SH ($n = 1275$)	P
ALBU (mg/L)	28.30 ± 51.54 ($n = 2047$)	24.31 ± 43.31 ($n = 1010$)	33.19 ± 59.91 ($n = 1037$)	<0.01
ALBU > 30 (mg/L)	383 (18.7)	150 (14.9)	233 (22.5)	<0.01
baPWV (cm/s)	1795.95 ± 396.67 ($n = 1742$)	1632.66 ± 310.95 ($n = 801$)	1934.95 ± 408.76 ($n = 941$)	<0.01
baPWV ≥ 1400 cm/s	1540 (88.4)	633 (79.0)	907 (96.4)	<0.01
IMT (mm)	0.92 ± 0.18 ($n = 2220$)	0.89 ± 0.16 ($n = 1064$)	0.94 ± 0.19 ($n = 1156$)	<0.01
IMT ≥ 1 mm	791 (35.6)	317 (29.8)	474 (41.0)	<0.01

Abbreviations: ALBU, microalbuminuria; baPWV, brachial–ankle pulse wave velocity; IMT, carotid intima–media thickness; SH, supine hypertension.

Table 3. Correlation analysis between different target organ indices and other variables.

	ALBU ($n = 2047$)		baPWV ($n = 1742$)		IMT ($n = 2220$)	
Variable	r	P	r	P	r	P
Age	0.04	0.07	0.32	<0.01	0.18	<0.01
Sex	−0.01	0.78	0.01	0.73	−0.15	<0.01
Supine SBP	0.14	<0.01	0.49	<0.01	0.16	<0.01
Sitting SBP	0.13	<0.01	0.25	<0.01	0.18	<0.01
Supine heart rate	0.11	<0.01	0.23	<0.01	0.02	0.03
BMI	0.10	<0.01	−0.01	0.75	0.06	0.06
FBG	0.16	<0.01	0.14	<0.01	0.01	0.65
TG	0.04	0.10	0.08	0.79	0.01	0.79
hsCRP	0.11	<0.01	0.01	0.98	0.06	0.02
LDL-C	0.01	0.58	−0.01	0.98	0.03	0.21
Smoking	0.01	0.71	−0.08	<0.01	0.06	0.01
Drinking	0.01	0.82	−0.03	0.23	0.03	0.13
Exercising	−0.05	0.04	−0.03	0.16	−0.04	0.04
Antihypertensive medications	0.07	<0.01	0.20	<0.01	−0.07	<0.01

Abbreviations: ALBU, microalbuminuria; baPWV, brachial-ankle pulse wave velocity; BMI, body mass index; FBG, fasting blood glucose; hsCRP, hypersensitive C-reactive protein; IMT, carotid intima–media thickness; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides.

with ALBU, baPW, and IMT ($P < 0.05$). FBG and the use of antihypertensive medications were positively correlated with ALBU and baPWV ($P < 0.01$). hsCRP levels were positively correlated with ALBU and IMT ($P < 0.05$). Age was positively correlated with baPWV and IMT (Table 3).

Multivariate logistic regression analysis of factors influencing target organ damage indices

Model 1 assumed ALBU, baPWV, IMT as dependent variables and SH as the independent variable. Nonconditional logistic regression analysis was carried out to determine the effect of SH on ALBU, baPWV, and IMT. The results indicated that SH was a risk factor for ALBU, baPWV, and increased IMT. Model 2 was based on Model 1, with corrections for age and sex (male/female). The results showed that SH was a risk factor for ALBU, baPWV, and increased IMT. Model 3 was based on Model 2, with corrections for sitting SBP, sitting DBP, supine heart rate, BMI, FBG, LDL-C, TG, hsCRP, smoking, alcohol consumption, exercise, and use of antihypertensive medications. The findings indicated that SH was a risk factor for baPWV and increased IMT ($P < 0.05$), but the correlation between SH and ALBU after correction for associated factors was not statistically significant. The odds ratios (OR) were 5.15 (95% CI: 3.43–7.73), 1.47 (95% CI: 1.17–1.87), and 1.34 (95% CI: 0.99–1.82), respectively (Table 4).

Discussion

We demonstrated an association between SH and the indices of target organ damage ALBU, baPWV, and IMT in a large sample of subjects aged over 60 years. During the measurement of supine blood pressure, individuals are relatively relaxed, which may largely reflect real blood pressures. In addition, the reduced venous return and relatively stable hormone levels observed in vivo in the supine position can lead to more accurate blood pressure measurements.[30] Garland et al.[14] studied 65 cases of elderly patients with autonomic failure and found

Table 4. Multivariate logistic regression analysis of factors influencing target organ damage indices.

	Model 1		Model 2		Model 3	
Variable	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
ALBU (mg/L)	1.66 (1.33–2.08)	<0.01	1.61 (1.28–2.03)	<0.01	1.34 (0.99–1.82)	0.06
baPWV (cm/s)	7.08 (4.83–10.4)	<0.01	6.76 (4.58–10.0)	<0.01	5.15 (3.43–7.73)	<0.01
IMT (mm)	1.64 (1.37–1.95)	<0.01	1.51 (1.25–1.81)	<0.01	1.47 (1.17–1.87)	<0.01

Abbreviations: ALBU, microalbuminuria; baPWV, brachial–ankle pulse wave velocity; BMI, body mass index; CI, confidence interval; FBG, fasting blood glucose; hsCRP, hypersensitive C-reactive protein; IMT, carotid artery intima–media thickness; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio; SBP, systolic blood pressure; SH, supine hypertension.

Model 1 assumed ALBU (<30 mg/L assigned 0, ≥ 30 mg/L assigned 1), baPWV (<1400 cm/s assigned 0, ≥ 1400 cm/s assigned 1), and IMT(<1. 00 mm assigned 0, ≥ 1. 00 mm assigned 1) as dependent variables, and SH (Yes/No) as the independent variable. Univariate regression analysis was carried out. Model 2 was based on Model 1, with correction for age and sex. Model 3 was based on Model 2, with correction for sitting SBP (<140 mmHg assigned 0, ≥ 140 mmHg assigned 1), sitting DBP (<90 mmHg assigned 0, ≥ 90 mmHg assigned 1), BMI (<24 kg/m^2 assigned 0, ≥ 24 kg/m^2 assigned 1), FBG (<6.1 mmol/L assigned 0, ≥ 6.1 mmol/L assigned 1), LDL-C (≤3.64 mmol/L assigned 0, > 3.64 mmol/L assigned 1), hsCRP (<2 mg/L assigned 0, ≥ 2 mg/L assigned 1), smoking (No/Yes), alcohol consumption (No/Yes), exercise (No/Yes), and use of antihypertensive medication (No/Yes).

that SH was related to reduced glomerular filtration rate. Cuspidi et al.[20] studied 343 untreated hypertension patients, among whom the number with left ventricular hypertrophy was three times greater in the nighttime SH group than in the group with normal nighttime blood pressure. However, these studies of the relationship between SH and target organ damage involved relatively small populations.

In the present study, the occurrence rate of target organ damage and the average values of ALBU, baPWV, and IMT were higher in the SH group than in the non-SH group, while supine SBP was positively correlated with ALBU, baPWV, and IMT. To date, most studies have focused on nighttime blood pressure in 24-h ambulatory blood pressure monitoring in relation to target organs,[10–12,20,21] whereas daytime supine blood pressure affecting target organ damage has been largely overlooked. Cuspidi et al.[20] found evidence that nighttime supine SBP in 343 hypertensive patients was positively correlated with IMT ($r = 0.22$, $P = 0.01$). Ozawa et al.[21] reported a positive linear correlation between nighttime supine SBP and baPWV ($\beta = 0.297$, $P < 0.01$) in elderly hypertensive patients. These results confirmed the strong correlation between supine SBP and target organ damage.

In addition, in the present study, multivariate logistic regression analysis showed that SH was a risk factor for target organ damage, as reflected by various indices. Sharabi et al.[22] observed that, in patients with autonomic failure, SH increased the risk of chronic renal failure, while de la Sierra et al.[23] reported that nighttime SH was a risk factor for positive urinary protein (OR = 1.021; 95% CI: 1.012–1.029). Chatzistamatiou et al.[24] studied 182 cases of untreated hypertensive patients and found that patients in the SH group had a higher average carotid–femoral pulse wave velocity (cfPWV) value and greater IMT compared with patients in the non-SH group (cfPWV: 8.6 ± 1.6 m/s vs. 7.9 ± 1.4 m/s, $P = 0.009$; IMT: 0.74 ± 0.17 mm vs. 0.68 ± 0.15 mm, $P = 0.007$). Androulakis et al.[25] studied 319 hypertensive patients and found that nighttime SH was strongly associated with target organ damage (cfPWV, IMT, and left ventricular hypertrophy). These results suggest that SH and target organ damage are closely related. Our study demonstrates that SH is closely associated with vascular function and structural changes, although the relationship between SH and renal damage remains to be further studied. Other studies have indicated that SH is a risk factor for kidney damage, left ventricular hypertrophy, and other target organ damage.[13–15] Kikuya et al.[26] followed 1332 healthy individuals over 10.8 years old and found that nighttime supine blood pressure was a better predictor of cardiovascular disease mortality than daytime blood pressure.

Our study has added to the evidence that SH has a relationship with the risk for target organ damage, given that sitting hypertension has previously been shown to be a risk factor for target organ damage. With an aging population and increased incidence of stroke in China,[31] our results can be used for evaluating the health of patients lying in bed. Because of the cross-sectional design of our study, we could not establish a causal relationship between SH and target organ damage. However, based on previous studies, we can infer that as supine blood pressure increases, hormone levels also change.[27] Hence, the sympathetic nervous system is stimulated, which leads to elevated levels of vasoconstrictor substances such as catecholamines, and vascular resistance increases in turn.[14] Consequently, glomerular perfusion decreases, renal dysfunction is aggravated, and the resulting abnormal leakage of protein may result in increased ALBU.

Furthermore, SH often occurs in patients with autonomic failure.[3,14] Previous studies[14] showed that, in these patients, the tension of the postganglionic sympathetic nerve increases, the regulatory function of the pressure sensor decreases significantly, and the arterial structure could also change. Changes in arterial structure are often presented as an increase in the wall-to-lumen ratio. Atrial wall thickening results in increased arterial stiffness, which can lead to increased baPWV and IMT. The inverse relationship should also be considered. ALBU is an independent risk factor for vascular damage,[28] and an increase in ALBU leads to increased arterial stiffness. Increases in baPWV and IMT also suggest increased arterial stiffness. As arterial stiffness increases, the regulatory function of the pressure sensors inside the artery wall will be affected, which in turn increases supine blood pressure.

Limitations

This study has some limitations. First, it was a cross-sectional study, so it could not establish a causal relationship between SH and target organ damage. However, very few previous studies have investigated the association between supine blood pressure and target organ damage in a large sample of the late middle-aged population. Second, our participants were predominantly male, which might have had an impact on our results. In addition, our results apply only to subjects over 60 years old. Further investigation will be required to determine whether our results can be extended to other population groups. Nevertheless, this study is instructive with respect to blood pressure management and the prevention of subclinical target organ damage in subjects over 60 years old.

Conclusions

Our study demonstrates that SH is a risk factor for target organ damage, as determined by the indices baPWV and IMT, in subjects over 60 years old. The association of SH with ALBU and kidney damage requires further study. While adding to our knowledge of the relationship between blood pressure and target organ damage, the study also underlines the importance of supine blood pressure in evaluating and possibly reducing subclinical target organ damage. However, further studies will be required to confirm the replicability of our results and to determine whether they can be extrapolated to a wider population.

Acknowledgements

We thank all participants and staff of the Kailuan study for their important contributions.

Declaration of conflicting interest

The Authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

1. Cicolini G, Pizzi C, Palma E, et al. Differences in blood pressure by body position (supine, Fowler's, and sitting) in hypertensive subjects. *Am J Hypertens* 2011; 24: 1073–1079.
2. Frewen J, Finucane C, Savva GM, et al. Orthostatic hypotension is associated with lower cognitive performance in adults aged 50 plus with supine hypertension. *J Gerontol A Biol Sci Med Sci* 2014; 69: 878–885.
3. Okamoto LE, Gamboa A, Shibao C, et al. Nocturnal blood pressure dipping in the hypertension of autonomic failure. *Hypertension* 2009; 53: 363–369.
4. Bliziotis IA, Destounis A and Stergiou GS. Home versus ambulatory and office blood pressure in predicting target organ damage in hypertension: a systematic review and meta-analysis. *J Hypertens* 2012; 30: 1289–1299.

5. Su TC, Lee YT, Chou S, et al. Twenty-four-hour ambulatory blood pressure and duration of hypertension as major determinants for intima-media thickness and atherosclerosis of carotid arteries. *Atherosclerosis* 2006; 184: 151–156.
6. Ushigome E, Fukui M, Hamaguchi M, et al. Maximum home systolic blood pressure is a useful indicator of arterial stiffness in patients with type 2 diabetes mellitus: post hoc analysis of a cross-sectional multicenter study. *Diabetes Res Clin Pract* 2014; 105: 344–351.
7. Wei FF, Li Y, Zhang L, et al. Beat-to-beat, reading-to-reading, and day-to-day blood pressure variability in relation to organ damage in untreated Chinese. *Hypertension* 2014; 63: 790–796.
8. Lewington S, Clarke R, Qizilbash N, et al. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet* 2002; 360: 1903–1913.
9. Matsui Y, Ishikawa J, Eguchi K, et al. Maximum value of home blood pressure: a novel indicator of target organ damage in hypertension. *Hypertension* 2011; 57: 1087–1093.
10. Milazzo V, Maule S, Di Stefano C, et al. Cardiac organ damage and arterial stiffness in autonomic failure: comparison with essential hypertension. *Hypertension* 2015; 66: 1168–1175.
11. Hara A, Tanaka K, Ohkubo T, et al. Ambulatory versus home versus clinic blood pressure: the association with subclinical cerebrovascular diseases: the Ohasama study. *Hypertension* 2012; 59: 22–28.
12. Wei FF, Li Y, Zhang L, et al. Association of target organ damage with 24-hour systolic and diastolic blood pressure levels and hypertension subtypes in untreated Chinese. *Hypertension* 2014; 63: 222–228.
13. Vagaonescu TD, Saadia D, Tuhrim S, et al. Hypertensive cardiovascular damage in patients with primary autonomic failure. *Lancet* 2000; 355: 725–726.
14. Garland EM, Gamboa A, Okamoto L, et al. Renal impairment of pure autonomic failure. *Hypertension* 2009; 54: 1057–1061.
15. Zuin M, Schiavon L, Ramazzina E, et al. 2C.03: Independent predictors of nocturnal hypertension in elderly subjects from general population: the risk of vascular complication, impact of genetic in old people (ROVIGO) study. *J Hypertens* 2015; 33(Suppl 1): e25.
16. Yu K, Xing A, Wang D, et al. Prevalence and relative risk factors of atrial fibrillation in male coal miners in North China. *Int J Cardiol* 2014; 174: 223–224.
17. Ogedegbe G, Spruill TM, Sarpong DF, et al. Correlates of isolated nocturnal hypertension and target organ damage in a population-based cohort of African Americans: the Jackson Heart Study. *Am J Hypertens* 2013; 26: 1011–1016.
18. Papaioannou TG, Protogerou AD, Nasothimiou EG, et al. Assessment of differences between repeated pulse wave velocity measurements in terms of 'bias' in the extrapolated cardiovascular risk and the classification of aortic stiffness: is a single PWV measurement enough? *J Hum Hypertens* 2012; 26: 594–602.
19. Hedblad B, Wikstrand J, Janzon L, et al. Low-dose metoprolol CR/XL and fluvastatin slow progression of carotid intima-media thickness: main results from the beta-blocker cholesterol-lowering asymptomatic plaque study (BCAPS). *Circulation* 2001; 103: 1721–1726.
20. Cuspidi C, Sala C, Valerio C, et al. Nocturnal hypertension and organ damage in dippers and nondippers. *Am J Hypertens* 2012; 25: 869–875.
21. Ozawa M, Tamura K, Okano Y, et al. Blood pressure variability as well as blood pressure level is important for left ventricular hypertrophy and brachial-ankle pulse wave velocity in hypertensives. *Clin Exp Hypertens* 2009; 31: 669–679.
22. Sharabi Y and Goldstein DS. Mechanisms of orthostatic hypotension and supine hypertension in Parkinson disease. *J Neurol Sci* 2011; 310: 123–128.
23. de la Sierra A, Gorostidi M, Banegas JR, et al. Nocturnal hypertension or nondipping: which is better associated with the cardiovascular risk profile? *Am J Hypertens* 2014; 27: 680–687.

24. Chatzistamatiou EI, Moustakas GN, Veioglanis S, et al. Nocturnal hypertension: poor correlation with office blood pressure but strong prognostic factor for target organ damage. *Hellenic J Cardiol* 2012; 53: 263–272.
25. Androulakis E, Papageorgiou N, Chatzistamatiou E, et al. Improving the detection of preclinical organ damage in newly diagnosed hypertension: nocturnal hypertension versus non-dipping pattern. *J Hum Hypertens* 2015; 29: 689–695.
26. Kikuya M, Ohkubo T, Asayama K, et al. Ambulatory blood pressure and 10-year risk of cardiovascular and noncardiovascular mortality: the Ohasama study. *Hypertension* 2005; 45: 240–245.
27. Goldstein DS, Pechnik S, Holmes C, et al. Association between supine hypertension and orthostatic hypotension in autonomic failure. *Hypertension* 2003; 42: 136–142.
28. Sung JK, Kim JY, Youn YJ, et al. Urine albumin creatinine ratio is associated with carotid atherosclerosis in a community based cohort: atherosclerosis risk of rural area in Korean general population study. *J Cardiovasc Ultrasound* 2010; 18: 134–138.
29. Perloff D, Grim C, Flack J, et al. Human blood pressure determination by sphygmomanometry. *Circulation* 1993; 88: 2460–2470.
30. Netea RT, Lenders JW, Smits P, et al. Influence of body and arm position on blood pressure readings: an overview. *J Hypertens* 2003; 21: 237–241.
31. Wang J, An Z, Li B, et al. Increasing stroke incidence and prevalence of risk factors in a low-income Chinese population. *Neurology* 2015; 84: 374–381.