Aortic Blood Pressure and Central Hemodynamics Measured by Noninvasive Pulse Wave Analysis in Gujarati Normotensives

Jayesh Dalpatbhai Solanki, Hemant B Mehta, Chinmay J Shah
Department of Physiology, Government Medical College, Bhavnagar, Gujarat, India

Abstract

Background and Aim: Central blood pressure (BP) and central hemodynamics are immediate and discrete parameters inferring about the cardiovascular system. They can be studied noninvasively by pulse wave analysis (PWA). Before use, these parameters need normative baseline study to set reference values. **Methods:** We conducted a cross-sectional study in 900 normotensives, aged 15–65 years (divided into five subgroups). Oscillometric PWA was accomplished by Mobil-O-Graph (IEM, Germany). Aortic BP, cardiac output (CO) and index, peripheral resistance, stroke volume and index, stroke work, difference between brachial and aortic systolic, and pulse pressure were the studied parameters. They were analyzed further with respect to subgroup based on age, gender, and body mass index (BMI). Multiple regressions were done to find significant predictors of central hemodynamics. $P < 0.05$ was taken as statistical significance. **Result:** There were five age-based subgroups from 15 to 65 years, showing increase in central BP- and CO-related parameters age. Males had significantly higher results than females except heart rate (HR) and peripheral resistance. BMI ≥23 was related to significantly higher results. Age, HR, height, weight, and BMI were not significant predictors of PWA parameters. Central hemodynamics were predicted by brachial BPs, systolic and diastolic more than mean or pulse. **Conclusion:** It is feasible to assess central hemodynamics by PWA. Age, gender, BMI, HR, and brachial pressure affect the central hemodynamics in normotensive individuals. These baseline data can be referred for the future studies in our population.

Keywords: Central blood pressure, central hemodynamic, normotensive, oscillometric, pulse wave analysis

Received: 09th April, 2018; ***Revised:*** 11th May, 2018; ***Accepted:*** 16th May, 2018

Introduction

Cardiovascular health has now become a global periodical need.[1] Cardiovascular diseases have now become the leading cause of mortality in India, responsible for a quarter of all mortality.[2] Brachial blood pressure (bBP) is a routine parameter, but with some limitations. Peripheral arterial pressure may be an inaccurate substitute for BP present in the central aorta.[3] Owing to pulse pressure amplification (PPA), aortic systolic BP (SBP) is usually lower in brachial SBP.[4] The heart and large arteries like the aorta are directly exposed to the central BP rather than the brachial/peripheral BP. Thus, central BP may potentially have a superior value for the prediction of cardiovascular events.[4] Similarly, cardiac output (CO) and stroke work (SW) are functionally most important outcome parameters of functioning of the heart. These parameters are not routinely measured and have not been studied owing to the invasive nature of available measurement tools.[5] However, with innovation in pulse wave analysis (PWA) and generalized transfer factor, now the same can be measured noninvasively. Mobil-O-graph is one such cuff-based tool that enabled measurement of discrete central hemodynamic parameters.[6-8] We previously published the utility of these parameters to underscore cardiovascular progeria in young individuals with a family history of diabetes or hypertension (HTN).[9,10] In light of lack of baseline normative data and potential of applying this tool for further work, we conducted this study on large sample of normotensive individuals. The current paper aimed to describe central hemodynamics in the same.

Access this article online	
Quick Response Code:	**Website:** www.ijcep.org
	DOI: 10.4103/ijcep.ijcep_24_18

Address for correspondence: Dr. Jayesh Dalpatbhai Solanki, F1, Shivganga Appartments, Plot No 164, Bhayani Ni Waadi, Opp. Bawaliya Hanuman Temple, Gadhechi Wadlaa Road, Bhavnagar - 364 001, Gujarat, India.
E-mail: drjaymin_83@yahoo.com

How to cite this article: Solanki JD, Mehta HB, Shah CJ. Aortic blood pressure and central hemodynamics measured by noninvasive pulse wave analysis in Gujarati normotensives. Int J Clin Exp Physiol 2018;5:75-80.

Materials and Methods

Study setup and study participants

A cross-sectional study was conducted at clinical research laboratory of physiology department of a government medical college affiliated to a tertiary care teaching government hospital from June 18, 2015 to February 3, 2018. The study protocol was first approved by the Institutional Review Board. The sample size was calculated by Raosoft software (Raosoft, Inc. free online software, Seattle, WA, USA). For 95% confidence level, 5% precision and response distribution 50%, a sample size of 900 was adequate for population of our city 6 lakhs. Using convenience sampling method, we enrolled 1257 apparently healthy individuals from our institute and community. After scrutiny, we finally had 900 individuals considered for this study.

Inclusion and exclusion criteria

We included apparently healthy nonathletic individuals, aged 15–65 years, of either sex, nonalcoholic, nonsmoking, not known to have any acute or chronic disease, not taking any medical treatment, and ready to give written informed consent. Apart from participants not fulfilling these criteria, we excluded participants using any alternative system of medicines/lifestyle managements such as Yoga and meditation and participants screened to have newly diagnosed HTN (n = 123). Three participants were excluded from the study after pulse wave recording, owing to irregular pulse rhythm. After recording, 16 readings were discarded due to the poor quality of pulse wave recordings. Seven patients were excluded with arm circumference beyond available cuff size. Hence, the final sample size was 900.

Subject assessment and definitions

All patients were personally interviewed for general features, demographic characteristics, and relevant history. SBP ≥140 mmHg and diastolic BP (DBP) ≥90 mmHg or use of antihypertensive medication was defined as HTN.

Instrument used-Mobil-O-graph

We used portable, PC-attached calibrated and validated[4] instrument Mobil-O-Graph (IEM Gmbh, Stolberg, Germany) owned by the physiology department to record brachial pulse wave. It contained three different sized arm cuffs, connecting tube, recorder, bluetooth, licensed software, and laptop. It performs PWA based on the oscillometric principle and analysis of pressure pulse wave. Initially, mid-arm circumference of the left arm is measured to choose the BP cuff of appropriate size – small (20–24 cm), medium (24–32 cm), or large (32–38 cm). It is wrapped around the left arm and tubing is connected to the recorder device as per standard protocol. As per the ARCSolver algorithm, a recording device generates pressure in the cuff by self-inflation and deflation follows it in a stepwise manner. If the first reading is free of artifact and error, there is a pause of 30 s to follow, after which there is second bout of inflation and deflation. During deflation, the cuff is kept inflated at brachial diastolic pressure for 10 s which allows intermittent flow that produces pressure pulse waves. Brachial arterial pulsation generates the pressure oscillations which are transmitted to BP cuff tied around the left arm and measured by transducer to be fed into microprocessor. Brachial artery pulse wave is recorded by the instrument and central aortic pulse wave is derived by validated a generalized. There are further point-based and area-based analyses by computer software to derive various cardiovascular parameters.

Measurement protocol[11]

A BP cuff of appropriate size was chosen based on measured mid-arm circumference and applied to the left arm using standard protocol. All readings were taken after 10 min of rest, in postabsorptive phase with participants avoiding smoking or alcohol for 12 h before the test, in a calm room avoiding external influences or arm movement. Measurements were taken twice in each participant. Owing to objective, algorithm-based, validated measurement protocol, there is good inter or intraobserver reproducibility.

Parameters measured

1. Heart rate (HR), body mass index (BMI), body surface area (BSA)
2. Brachial BP (bBP) – systolic (bSBP), diastolic (bDBP), pulse (bPP), and mean (bMBP)
3. Central BP (cBP) – systolic (cSBP), diastolic (cDBP), and pulse (cPP)
4. Central hemodynamics – CO, cardiac index (CI), systemic vascular resistance.

Parameters derived[9]

1. Stroke volume (SV) – CO/HR
2. SV index (SVI) – SV/BSA
3. SW – (pulse pressure [PP]) × (SV) × 0.0144
4. PPA – brachial PP/aortic PP
5. ΔSBP = difference between brachial and aortic SBP
6. ΔPP = difference between brachial and aortic DBP.

Statistical analysis of data

Collected data were entered into Excel spreadsheet and descriptive analysis was expressed as mean ± standard deviation until indicated specifically. All statistical calculations were done by GraphPad InStat 3 software (demo version free software of GraphPad Software, Inc. California, USA) and MedCalc Statistical Software version 16.4.3 (MedCalc Software bvba, Ostend, Belgium; https://www.medcalc.org; 2016). We calculated the statistical significance of differences of various numerical parameters between various groups by Mann–Whitney test or unpaired Student t-test for two groups and by ANOVA test for more than two groups. Multiple linear regressions were used to find major and significant predictors of main study outcomes. Statistical significance level was set at $P < 0.05$.

Results

We studied PWA-based central hemodynamics of 900 nonhypertensive participants (541 males and 359 females) in age-based subgroups – 15–24, 25–34, 35–44, 45–54, and

55–65 years. Five Groups from A to E were comparable for gender distribution, height, weight, BMI, and HR. Central BP- and CO-related parameters were showing statistically significant and increasing trends with age. However, PPA, ΔSBP, and ΔPP showed decreasing trend with significant *P* values. In most *post hoc* tests of intergroup caparisons, Group A had statistically significant difference with any other group in most instances [Table 1].

Gender-based subgroups had comparable age, BMI, cPP, and CI. Males had significantly higher height, weight, cSBP, cDBP, and CO-related parameters than females. Females had significantly higher HR and PR than males. PPA, ΔSBP, and ΔPP were not significantly different between the two groups.

Using cutoff 23, BMI-based subgroups were compared. Group with BMI ≥23 had significantly higher central BP, CO, SW, and SV but significantly lower PR, CI, SVI, PPA, ΔSBP, and ΔPP as compared to group with BMI <23 [Table 2].

By multiple linear regression models, we tested predictors of central hemodynamics (dependant parameters) from independent parameters. Age (except CO and SW) and HR (except cDBP) were major consistent significant predictors. Height, weight, and BMI were insignificant predictors of most study parameters. Most study parameters were significantly predicted by bBP, more by systolic, diastolic than mean, and PP [Table 3].

Table 1: Age- and gender-wise distribution of baseline data and central hemodynamics (mean and standard deviation) in the study group (*n*=900)

Parameter, unit	(A) 15-24 years (n=226)	(B) 25-34 years (*n*=174)	(C) 35-44 years (*n*=192)	(D) 45-54 years (*n*=215)	(E) 55-65 years (*n*=93)	Significant *P* *Post hoc* test in subgroups A to E
Male (%)	143 (63)	109 (63)	100 (52)	136 (76)	53 (57)	0.10
Height (cm)	164.43 (9.76)	164.52 (12.27)	162.86 (7.33)	162.49 (6.50)	162.95 (6.79)	0.0374* None
Weight (kg)	57.01 (12.63)	63.20 (11.68)	63.75 (9.98)	63.37 (9.68)	60.08 (10.05)	<0.0001* A/B, A/C, A/D
BMI (kg/m²)	20.92 (3.62)	23.47 (4.20)	24.02 (3.45)	23.94 (3.41)	22.45 (2.49)	<0.0001* A/B, A/C, A/D, A/E
HR (beats/min)	89.96 (12.90)	88.98 (14.50)	92.12 (14.64)	86.63 (12.90)	85.92 (13.28)	0.0001* None
cSBP (mm Hg)	108.96 (10.39)	111.08 (10.05)	113.29 (15.31)	115.89 (12.54)	123.77 (17.14)	<0.0001* A/C, A/E, A/D
cDBP (mm Hg)	80.07 (8.64)	80.00 (10.28)	82.71 (11.57)	84.87 (11.02)	87.66 (10.28)	<0.0001* A/E, A/D
cPP (mm Hg)	28.81 (7.53)	30.87 (7.48)	31 (8.9)	31.07 (8.31)	35.67 (12.45)	<0.0001* A/E
CO (L/min)	4.73 (0.59)	4.73 (0.55)	4.83 (0.66)	4.80 (0.61)	5.01 (0.73)	0.0203* A/E
PR (mm Hg/mL)	1.24 (0.14)	1.25 (0.13)	1.25 (0.12)	1.28 (0.13)	1.31 (0.16)	0.0276* None
CI (L/min/m²)	2.97 (0.38)	2.82 (0.35)	2.88 (0.42)	2.87 (0.40)	3.05 (0.49)	<0.0001* A/B, A/C, A/D
SV (ml/beat)	53.59 (25.77)	54.61 (11.57)	53.66 (10.42)	56.46 (10.12)	59.16 (9.63)	<0.0001* A/E, A/D
SVI (ml/m²/beat)	33.70 (6.33)	32.52 (6.69)	32.02 (6.55)	33.77 (6.52)	36.09 (5.93)	<0.0001* A/C, A/E
SW (g m/beat)	94.42 (23.35)	95.33 (23.30)	95.37 (26.33)	101.93 (25.43)	114.35 (30.83)	<0.0001* A/D, A/E
ΔSBP (mmHg)	12.35 (4.93)	9.59 (3.91)	8.82 (7.47)	8.40 (3.40)	8.77 (4.08)	<0.0001* All
ΔPP (mmHg)	14.37 (5.69)	11.44 (5.92)	9.79 (4.43)	9.8 (4.03)	10.44 (4.02)	<0.0001* All
PPA	1.51 (0.20)	1.38 (0.22)	1.33 (0.16)	1.33 (0.13)	1.32 (0.13)	<0.0001* All

*Represents comparison with A. *P<0.05; **P<0.01; ***P<0.001. HR: Heart rate, cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cMBP: Central mean blood pressure, PP: Pulse pressure, cPP: Central PP, CO: Cardiac output, PR: Peripheral resistance, CI: Cardiac index, SV:Stroke volume, SVI: Stroke volume index, SW: Stroke work, SBP: Systolic blood pressure, ΔSBP: Difference of brachial and central SBP, ΔPP: Difference between brachial and central PP, PPA: PP amplification, BMI: Body mass index

Table 2: Gender- and body mass index (cutoff 23)-wise distribution of baseline data and central hemodynamics (mean and standard deviation) in the study group (n=900)

Parameter, unit	Males (n=541)	Females (n=359)	P	BMI <23 (n=468)	BMI ≥23 (n=432)	P
Age (years)	36.26±13.39	37.04±13.04	0.45	34.05±13.96	39.30±11.85	<0.0001*
Male, *n* (%)	-	-	-	277 (59)	264 (61)	0.59
Height (cm)	166.58±8.47	158.84±7.48	<0.0001*	163.42±9.49	163.60±8.34	0.10
Weight (kg)	63.65±11.17	58.21±10.71	<0.0001*	54.05±7.54	69.53±8.94	<0.0001*
BMI (kg/m^2)	22.91±3.68	23.02±3.90	0.93	20.20±1.98	25.94±2.87	<0.0001*
HR (beats/min)	87.70±14.39	91.02±12.59	<0.0001*	88.32±13.12	89.78±14.46	0.19
cSBP (mmHg)	114.32±13.45	112.22±13.51	0.0018*	111.12±12.42	116.04±14.17	<0.0001*
cDBP (mmHg)	83.42±10.99	81.50±10.91	0.0038*	81.05±10.70	84.40±11.05	<0.0001*
cPP (mmHg)	30.99±8.43	30.75±8.97	0.33	29.99±8.44	31.88±8.76	0.0002*
CO (L/min)	4.93±0.63	4.61±0.56	<0.0001*	4.68±0.59	4.93±0.63	<0.0001*
PR (mm Hg/mL)	1.24±0.14	1.29±0.12	<0.0001*	1.27±0.12	1.26±0.14	0.0267*
CI (L/min/m^2)	2.90±0.40	2.92±0.41	0.55	3.01±0.40	2.79±0.38	<0.0001*
SV (ml/beat)	57.45±10.93	51.46±8.73	<0.0001*	54.13±10.67	56.08±10.28	0.0013*
SVI (ml/m^2/beat)	33.97±7.00	32.49±5.69	0.0071*	34.82±6.80	31.82±5.88	<0.0001*
SW (g m/beat)	103.84±25.74	90.83±24.44	<0.0001*	95.23±25.05	102.36±26.54	<0.0001*
ΔSBP (mmHg)	10.37±5.96	8.81±3.83	<0.0001*	9.98±4.39	9.50±6.08	0.0151*
ΔPP (mmHg)	11.90±5.68	10.41±4.20	0.0001*	11.77±5.36	10.80±4.96	0.0042*
PPA	1.40±0.21	1.36±0.16	0.0012*	1.41±0.21	1.35±0.16	<0.0001*

*Represents comparison with A. *P<0.05; **P<0.01; ***P<0.001. HR: Heart rate, cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, cMBP: Central mean blood pressure, PP: Pulse pressure, cPP: Central PP, CO: Cardiac output, PR: Peripheral resistance, CI: Cardiac index, SV: Stroke volume, SVI: Stroke volume index, SW: Stroke work, ΔSBP: Difference of brachial and central SBP, ΔPP: Difference between brachial and central PP, PPA: PP amplification

Table 3: Predictors of central hemodynamics by multiple linear regressions in the study group

Parameter	Statistic (*P*)	cSBP	cDBP	cPP	CO	SW	PPA
Age	*r* (partial)	0.25	−0.07	0.35	0.02	0.04	−0.34
	P	(<0.0001*)	(0.0439*)	(<0.0001*)	(0.61)	(0.28)	(<0.0001*)
Height	*r* (partial)	−0.04	−0.00	−0.06	0.02	0.03	0.07
	P	(0.26)	(0.08)	(0.10)	(0.57)	(0.36)	(0.0372*)
Weight	*r* (partial)	−0.01	0.04	0.01	0.02	0.01	−0.02
	P	(0.94)	(0.21)	(0.83)	(0.47)	(0.81)	(0.58)
BMI	*r* (partial)	0.03	0.00	0.00	−0.03	−0.01	0.01
	P	(0.42)	(0.98)	(0.92)	(0.35)	(0.85)	(0.87)
bSBP	*r* (partial)	0.39	0.36	0.30	0.17	0.17	-0.33
	P	(<0.0001*)	(<0.0001*)	(<0.0001*)	(<0.0001*)	(<0.0001*)	(<0.0001*)
bDBP	*r* (partial)	0.11	0.66	−0.29	−0.05	0.14	0.32
	P	(0.0009*)	(<0.0001*)	(<0.0001*)	(0.16)	(<0.0001*)	(<0.0001*)
bMBP	*r* (partial)	0.03	0.15	−0.04	0.01	0.03	0.06
	P	(0.30)	(<0.0001*)	(0.24)	(0.68)	(0.35)	(0.07)
bPP	*r* (partial)	−0.02	−0.37	0.19	0.02	0.23	0.37
	P	(0.55)	(<0.0001*)	(<0.0001*)	(0.47)	(<0.0001*)	(<0.0001*)
HR	*r* (partial)	−0.14	0.01	−0.18	0.27	−0.81	0.20
	P	(<0.0001*)	(0.86)	(<0.0001*)	(<0.0001*)	(<0.0001*)	(<0.0001*)

*Indicates statistical significance. b=Brachial, HR: Heart rate, cSBP: Central systolic blood pressure, cDBP: Central diastolic blood pressure, PP: Pulse pressure, cPP: Central PP, CO: Cardiac output, CI: Cardiac index, SW=Stroke work, SBP: Systolic blood pressure, PPA: PP amplification, BMI: Body mass index

Discussion

Cardiovascular health is the determinant of overall health and well-being.[12] Before using these validated, novel tools for epidemiological studies, it is important to set normative data. We did the same in our population using Mobil-O-graph that works on the oscillometric principle excluding the presence of cardiovascular disease, smoking, and diabetes.

All parameters showed a trend of significant increase with age which also was a major predictor for all. This result is in line with Nunan *et al.*[11] PP amplification with greater SBP and PP at brachial than central artery are known[13] and we found the same. Across all age groups, young to middle aged, a difference around 5–10 mmHg was found between bSBP-aSBP and bPP-aPP. Thus, adding central BP is beneficial. Gender, age, and obesity all are known to have a significant impact on PP amplification[13] and we found the same. It is suggested in young individuals, but it was evident even in 45–54 and 55–65 age groups. Cardiovascular aging produces accelerated central hemodynamics, so age must be considered as a major confounder while interpreting these results. Such difference is due to the underlying difference of arterial structure that is elastic aorta versus muscular peripheral arteries.[14] This method, being objective, adds further to the superiority than bBP which is objectively measured. It highly reliable as it is calibrated against intra-operatively measured direct invasive aortic BP. Males had higher central BP and CO-related parameters than females, in line with a previous study.[11] It is due to mean age which is in premenopausal range in most with advantage of female sex hormones.[15] However, it reverses in postmenopausal age group.[16] BMI is always a parameter affecting cardiovascular health[17] and the same was the trend in our study; BMI, though not being a significant predictor, was affecting result adversely with cutoff 23.[18] It supports the use of lower BMI cutoff in Asian adults and worse cardiovascular profile even at low adiposity level than non-asian population.[18] It is a preventable risk factor, so always a potential remains to improve cardiovascular health by reducing body weight to optimum.[19] Hence, age, gender, and BMI are confounders that must be accounted for while studying PWA central hemodynamics.

Most parameters were predicted by age and HR, in line with a known study.[11,20] However, we contrastingly find that height, weight, and BMI are not the predictors for central hemodynamics. Most parameters were predicted by bBP and that is in accordance with a previous study.[11] Yet, they add an advantage to fill the cardiovascular risk stratum, beyond bBP, owing to its immediate relation to the heart.[21]

The modern era is full of innovative techniques like PWA. Cardiovascular health is a state beyond blood pressure measured from peripheral arteries and HR. Better understanding by noninvasive, software-based, validated, calibrated, reproducible, objective, and cost-effective tool like Mobil-O-graph is there to be utilized. The present study was a baseline work on which further work can be mounted, and this normative data can be used to compare with cases in case-control studies involving various cardiovascular pathologies such as HTN, heart failure, diabetes, and atherosclerosis.

Limitations of the study

There were few limitations of our study such as exclusion of elderly patients and smokers and patients with diabetes and hypertensives; cross-sectional nature with no vertical follow-up; lack of biochemical investigations; and dependence of results on generalized transfer factor for central pulse wave derivations.

Conclusion

It is feasible to assess central hemodynamics by cuff-based oscillometric pressure PWA. Age, gender, BMI, HR, and brachial pressure affect the central hemodynamics in normotensive individuals. These are novel, objective, and direct parameters and these baseline data can be referred for the future studies in our population.

Acknowledgment

We are thankful to physiology department of our medical college for giving the facilities available in the department and to volunteers for participation in this study.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Brant LC, Ribeiro AL. Cardiovascular health: A global primordial need. Heart 2018. pii: heartjnl-2017-312562.
2. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: Current epidemiology and future directions. Circulation 2016;133:1605-20.
3. Alfonso M, Cymberknop L, Armentano R, Pessana F, Wray S, Legnani W. Arterial pulse pressure amplification described by means of a nonlinear wave model: Characterization of human aging. Int J Phys Conf Ser 2016;705:012029.
4. Kollias A, Lagou S, Zeniodi ME, Boubouchairopoulou N, Stergiou GS. Association of central versus brachial blood pressure with target organ damage: Systematic review and meta-analysis. Hypertension 2016;67:183-90.
5. Townsend RR, Wilkinson IB, Schiffrin EL, Avolio AP, Chirinos JA, Cockcroft JR, *et al.* Recommendations for improving and standardizing vascular research on arterial stiffness: A scientific statement from the American Heart Association. Hypertension 2015;66:698-722.
6. Wassertheurer S, Kropf J, Weber T, van der Giet M, Baulmann J, Ammer M, *et al.* A new oscillometric method for pulse wave analysis: Comparison with a common tonometric method. J Hum Hypertens 2010;24:498-504.
7. Weiss W, Gohlisch C, Harsch-Gladisch C, Tölle M, Zidek W, van der Giet M, *et al.* Oscillometric estimation of central blood pressure: Validation of the Mobil-O-Graph in comparison with the SphygmoCor device. Blood Press Monit 2012;17:128-31.
8. Weber T, Wassertheurer S, Rammer M, Maurer E, Hametner B, Mayer CC, *et al.* Validation of a brachial cuff-based method for estimating central systolic blood pressure. Hypertension 2011;58:825-32.
9. Solanki JD, Mehta HB, Shah CJ. Pulse wave analyzed cardiovascular parameters in young first degree relatives of type 2 diabetics – A cross-sectional study. Indian Heart J 2018;70:341-5.
10. Solanki JD, Mehta HB, Shah CJ. Pulse wave analysed cardiovascular parameters in young first degree relatives of hypertensives – A case control study. J Res Med Sci 2018;Ahead of print.
11. Nunan D, Wassertheurer S, Lasserson D, Hametner B, Fleming S, Ward A, *et al.* Assessment of central hemomodynamics from a brachial cuff in a community setting. BMC Cardiovasc Disord 2012;12:48.
12. Kanaya AM. India's call to action-prioritize chronic cardiovascular disease. JAMA Intern Med 2018;178:373-4.
13. Pichler G, Martinez F, Vicente A, Solaz E, Calaforra O, Redon J, *et al.* Pulse pressure amplification and its determinants. Blood Press 2016;25:21-7.
14. Lacolley P, Regnault V, Segers P, Laurent S. Vascular smooth muscle cells and arterial stiffening: Relevance in development, aging, and disease. Physiol Rev 2017;97:1555-617.
15. Shepherd J, Harvey R, Ranadive S, Miller V, Joyner M, Barnes J. Aortic pulse wave characteristics in postmenopausal women with and without a history of hypertensive pregnancy. FASEB J 2015;29 1 Suppl: 1053-6.
16. Harvey RE, Johnson MC, Ranadive SM, Joyner MJ, Lahr BD,

Miller VM, *et al*. Aortic hemodynamics in postmenopausal women following cessation of hormone therapy. Physiol Rep 2017;23:e13535.

17. Pichler G, Martinez F, Vicente A, Solaz E, Calaforra O, Lurbe E, *et al.* Influence of obesity in central blood pressure. J Hypertens 2015;33:308-13.
18. Misra A. Ethnic-specific criteria for classification of body mass index: A perspective for Asian Indians and American Diabetes Association position statement. Diabetes Technol Ther 2015;17:667-71.
19. Rankinen T, Sarzynski MA, Ghosh S, Bouchard C. Are there genetic paths common to obesity, cardiovascular disease outcomes, and cardiovascular risk factors? Circ Res 2015;116:909-22.
20. Yannoutsos A, Rinaldi ER, Zhang Y, Protogerou AD, Safar ME, Blacher J, *et al.* Central hemodynamics in risk assessment strategies: Additive value over and above brachial blood pressure. Curr Pharm Des 2015;21:719-29.
21. Rinaldi ER, Yannoutsos A, Borghi C, Safar ME, Blacher J. Central hemodynamics for risk reduction strategies: Additive value over and above brachial blood pressure. Curr Pharm Des 2015;21:730-6.