Original Article

Central hemodynamics and arterial stiffness by oscillometric pulse-wave analysis in treated Gujarati euglycemic hypertensives: A case-control study

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Abstract

Introduction: Hypertension is the most prevalent noncommunicable disorder, studied in terms of brachial blood pressure. Direct parameters like central hemodynamics and arterial stiffness, though superior, are not studied much. The same can be studied by pulse-wave analysis (PWA) and we did that in euglycemic treated hypertensives. **Materials and Methods:** A case-control study was conducted in 258 treated euglycemic hypertensives and 258 matched controls. Oscillometric PWA was accomplished by Mobil-O-Graph (IEM, Germany). Parameters were further analyzed for the effect of gender, physical activity, body mass index (BMI) (cutoff 23), blood pressure control, and duration (cutoff 5 years). Multiple linear regressions were used to find significant predictors. $P < 0.05$ was taken as statistically significant. **Results:** Cases had significantly higher brachial arterial parameters (blood pressure, heart rate, rate pressure product), arterial stiffness (augmentation pressure, augmentation index, pulse-wave velocity, total arterial stiffness, pulse pressure amplification), and central hemodynamics (central blood pressure, cardiac output, stroke work) compared to age, gender, and BMI-matched controls. In the case group, female gender, BMI ≥ 23, and uncontrolled blood pressure were significant factors affecting results. Heart rate and pulse pressure were major predictors of study parameters. Central pressure parameters were not predicted significantly by corresponding brachial pressure parameters. **Conclusion:** PWA revealed the adverse profile of arterial stiffness and central hemodynamics in treated Gujarati hypertensives, associated with female gender, BMI, and blood pressure control, predicted mainly by heart rate and pulse pressure, independent of brachial blood pressure. It indicates both potential and further study of these parameters.

Keywords: Arterial stiffness, blood pressure, hemodynamic, hypertensive, pulse-wave analysis

Introduction

India has witnessed an epidemiological transition in disease burden and deaths, with a steady rise in noncommunicable disease burden like systemic hypertension.[1] There are few limitations of routinely measured brachial blood pressure (bBP).[2] It can be rectified by the measurement of central hemodynamics and arterial stiffness which are key parameters in the context of the disease, yet not measured routinely.[3] Central blood pressure (cBP) is superior to bBP.[4] Central hemodynamics like cardiac output (CO) adds significantly to the details about the ultimate heart pumping. Arterial stiffness, similarly, is a gold standard in hypertension and measured in forms of pulse-wave velocity and augmentation index.[5] These stiffness and central hemodynamic parameters lack studies owing to lesser availability of instruments.[5] But instruments like Mobil-O-Graph are now available which performs oscillometric pressure pulse-wave analysis (PWA) noninvasively. This calibrated and validated device

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allows objective measurement of direct parameters like arterial stiffness and central hemodynamics, which are more important in hypertensives.[6-8] With this background, we conducted a Mobil-O-graph-based PWA study in treated hypertensives without diabetes.

Materials and Methods

Study design

It was a case-control study conducted on outdoor patients attending medicine and general outdoor patient department (OPD) of a tertiary care-teaching government hospital attached to a government medical college under the guidance of Physiology and Medicine departments from June 18, 2015 to March 02, 2018. Our study protocol was approved by the institutional review board of our college first.

Inclusion and exclusion criteria

We included apparently healthy, nonathletic hypertensives, taking antihypertensives regularly for at least 1 year, aged 15–65 years, of either sex, nonsmoking, nonalcoholic, not known for any acute or chronic systemic disease, ready to give written consent. Apart from noncompliance with these criteria, we excluded subjects doing any of the alternative systems of medicines/lifestyle managements like yoga and meditation.

Study groups

The sample size was calculated by Raosoft software (Raosoft, Inc. free online software, Seattle, WA, USA). To have 95% confidence level, 5% precision, considering response distribution 33%, a sample size of 474 was adequate for the population of the city (6 lakhs).

We screened and enrolled 700 hypertensives from general medicine outdoor patient department by simple random sampling. Out of these, we excluded 217 subjects with concomitant diabetes, 140 new hypertensives (duration less than 1 year), 68 due to history of irregular treatment, 10 due to the use of lifestyle modification, 3 due to irregular pulse-wave recording, 2 due to morbid obesity, and 2 owing to arm circumference beyond available cuff size. So, the case group finally had 258 euglycemic hypertensives.

To compare with, we selected 258 apparently healthy normotensive subjects from the pool of 1226 healthy controls who were matched head to head to the case group by age, gender, and body mass index (BMI).

Subject assessment and definitions

All subjects were personally interviewed in the form of questionnaires including general features, demographic characteristics, risk factor, self-reported moderate physical activity, and relevant disease history. A detailed history of pharmacotherapy used was elicited from each hypertensive, and regularity was confirmed by the patient's case report chart. Systolic blood pressure (SBP) ≥140 mmHg and diastolic blood pressure (DBP) ≥90 mmHg or use of antihypertensive medication was defined as hypertension. SBP <140 mmHg and DBP <90 mmHg were taken as blood pressure control.

Instrument used

We used portable, personal computer attached, calibrated,[7] and validated[8] instrument Mobil-O-Graph (IEM Gmbh, Stolberg, Germany) of the Physiology department to record brachial pulse wave. It undergoes oscillometric pressure PWA as per protocol designed by European Society of Hypertension (ESH).

Pressure oscillations are generated by brachial arterial pulsation, which is transmitted to bBP cuff and measured by the transducer to be fed into the microprocessor. Computerized software records pulse wave of the brachial artery and by validated a generalized transfer factor derives central aortic pulse wave. It further undergoes point-based and area-based analysis by computer to derive various cardiovascular parameters.

Measurement protocol

A blood pressure cuff of appropriate size (mid-arm circumference: 20–24 cm = small size, 24–32 cm = medium size, 32–38 cm = large size) was chosen based on measured mid-arm circumference and applied to left arm using a standard protocol. All readings were taken after resting for 10 min, in the postabsorptive phase while subjects avoiding smoking or alcohol for 12 h before measurement, in a calm room without external influences or avoiding arm movement.[7]

Parameters measured

As we used in the previous study, these are as follows[9]:

(1) Heart rate (HR), BMI, and body surface area (BSA)
(2) bBP – systolic, diastolic, pulse (bPP), and mean (bMBP)
(3) cBP – systolic (cSBP), diastolic (cDBP), and pulse (cPP)
(4) Central hemodynamics – CO, cardiac index, and systemic vascular resistance
(5) Arterial stiffness – augmentation pressure (AP), augmentation index at heart rate 75 per minute, reflection magnitude percentage (Ref%), and aortic pulse-wave velocity (aPWV)

Parameters derived

As we used in the previous study, these are as follows[9]:

(1) Rate pressure product (RPP) − (heart rate per minute) × (systolic blood pressure) × 10^{-2}
(2) Stroke volume − cardiac output/heart rate
(3) Stroke volume index − stroke volume/body surface area
(4) Stroke work (SW) − (pulse pressure) × (stroke volume) × 0.0144
(5) Total arterial stiffness − pulse pressure/stroke volume
(6) Pulse pressure index (PPI) − pulse pressure/systolic blood pressure
(7) Pulse pressure amplification (PPA) − brachial pulse pressure/aortic pulse pressure.

Statistical analysis

The data were entered to and sorted by Excel spreadsheet; numerical data were expressed as mean ± standard deviation until indicated specifically and qualitative data were expressed as number (%). Statistical calculations were done on 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; 2018). Numerical data were compared by the difference in mean/median distributions by unpaired *t*-test or Mann–Whitney test, based on results of Normality test for parametric distribution. We compared the distribution of qualitative data by Normality test or Chi-square test. Multiple linear regressions were used to find major and significant predictors of main study outcomes – central hemodynamics and arterial stiffness. The statistical significance level was taken as P value < 0.05.

Results

Case group of euglycemic-treated hypertensives (n = 258) and control group of matched normotensive controls (n = 258) had comparable mean age, weight, BMI, physical activity status, and gender distribution. Cases were significantly shorter than controls. Most study parameters including brachial blood pressures, RPP, vascular stiffness, and central hemodynamic parameters were higher in the case than the control with evident statistical significance for all except heart rate, reflection magnitude, PPA, and peripheral resistance. With cPP cutoff 40, cases had odds risk of 2.81 compared to controls with statistical significance (P < 0.0001) [Table 1]. In the case group, we compared males (n = 120) and female (n = 138). These subgroups were comparable for age, BMI, blood pressure control, use of pharmacotherapy, heart rate, RPP, brachial blood pressures, central blood pressure (systolic and diastolic), and central hemodynamics. Females had significantly shorter stature, lesser BMI, BSA, and prevalence of physical activity than males. Parameters of arterial stiffness were significantly higher in females than males except for pulse wave velocity, which was insignificantly higher in males. With cPP cutoff 40, females had odds risk of 1.78 compared to males with statistical significance (P = 0.054) [Table 1].

Physically active cases had no significantly different profile of PWA parameters than matched and comparable physically inactive cases. As compared to cases with BMI < 23, cases with BMI ≥ 23 had higher values of PWA parameters but statistical significance was not evident for all parameters. Odds risk for cPP ≥ 40 was 2.58 in cases with BMI ≥ 23 than those with BMI < 23 (P = 0.0062) [Table 2].

Blood pressure uncontrolled group had higher values of central blood pressure, central hemodynamics, and arterial stiffness than blood pressure controlled group, both groups being comparable for other parameters. Statistical significance was present for most of these differences except for heart rate, PPI, peripheral resistance, and most arterial stiffness parameters (except PWV). Cases with longer disease duration (≥5 years) had no significantly different profile of study parameters as compared to those with shorter disease duration (<5 years) [Table 3].

Using multiple linear regression models, we tested predictors of major PWA parameters (dependent parameters) of independent study parameters. Heart rate (positive for AP, cSBP, cPP, and SW and negative for rest) and brachial pulse pressure (positive for all except cDBP) were the major predictors of dependent parameters of arterials stiffness and central hemodynamics. Age was a major positive predictor only for PWV; bMBP was a significant positive predictor of cSBP and cDBP. Most central blood pressures were not significantly predicted by corresponding brachial blood pressure parameters [Table 4].

Discussion

The present study is by far the first study using Mobil-O-graph in middle-aged urban Indian hypertensives. PWA with generalized transfer factor gives parameters of cardiovascular health and aging, inferring beyond subjectively measured routine brachial blood pressure.[10] We excluded diabetics which add another dimension to cardiovascular aging and risk in hypertensives.[11,12] This gives us a chance to evaluate the effect of hypertension not due to hyperglycemia as it coincides in more than half of our hypertensives, and with the coexistence of the two, it is difficult to point out the effect of hypertension *per se* independently.[13,14] We compared treated hypertensives with age, gender, and BMI matched controls. Cases, being diagnosed and treated for at least 1 year, gave a chance to document the effect of disease on PWA-derived parameters after allowing the adequate benefit of blood pressure lowering and various other correlates for it.[15]

Cases had higher brachial, central hemodynamics and arterial stiffness than controls despite antihypertensive therapy for at least 1 year, comparable heart rate, BMI, age, and gender. Such results are supported by studies done elsewhere.[16] It can also be due to the higher prevalence of physical inactivity, poor blood pressure control, ethnic predisposition, delayed diagnosis, and lack of lifestyle modification. There is a link between aging, arterial stiffness, hypertension, and cardiovascular risk,[17] and the same is hinted even in treated individuals. We found PPA, PPI, and peripheral resistance insignificantly different as these parameters are from peripheral arteries which are better controlled by antihypertensives. It also highlights the importance of aortic and central parameters. This accelerated profile indicates the increase in workload on heart that can produce an adverse effect on heart as well as other organs downstream.[18]

Females had significantly higher values of bPP, cPP, RPP, and arterial stiffness except for PWV than males, in line with the previous study.[19] HR (determined by vagal tone), PWV (an exclusive function of age), and cDBP (related to arterioles

Table 1: Compassion of baseline and study parameters between cases and matched controls and male cases versus female cases

Parameter (unit)	Cases (*n*=258)	Controls (*n*=258)	*P*	Male cases (*n*=120)	Female cases (*n*=138)	*P*
Age (years)	48.68±7.60	49.00±8.23	0.56	49.62±6.85	47.87±8.13	0.10
Male, no (%)	120 (47%)	120 (47%)	1.00	–	–	–
Height (cm)	160.59±5.74	162.18±6.93	**<0.0001**	163.27±4.44	158.25±5.74	**<0.0001***
Weight (kg)	63.29±10.52	63.90±9.96	0.31	64.93±9.85	61.86±10.89	**0.0088***
BMI (kg/m^2)	24.47±3.45	24.23±3.27	0.33	24.30±3.31	24.62±3.58	0.45
BSA (m^2)	1.68±0.16	1.69±0.15	0.34	1.71±0.15	1.65±0.17	**0.0019***
P A, no (%)	51 (20%)	54 (21%)	0.85	36 (30%)	15 (11%)	**0.0001***
Duration (years)	4.88±3.75	–	–	4.21±2.99	5.45±4.22	**0.0452***
BPC, no (%)	98 (38%)	–	–	50 (42%)	48 (35%)	0.30
Drugs use						
ACEI, no (%)	173 (67%)	–	–	86 (72%)	87 (63%)	0.15
BB, no (%)	128 (50%)	–	–	67 (56%)	61 (44%)	0.08
CCB, no (%)	97 (38%)	–	–	41 (34%)	56 (41%)	0.31
Diuretics, no (%)	37 (14%)	–	–	14 (12%)	23 (17%)	0.29
ARB, no (%)	44 (17%)	–	–	22 (18%)	22 (16%)	0.62
Aspirin, no (%)	60 (23%)	–	–	41 (34%)	35 (25%)	0.13
Statin, no (%)	76 (29%)	–	–	36 (30%)	24 (17%)	**0.0186***
bBP (mmHg)						
SBP	137.79±19.14	126.67±15.94	**<0.0001***	137.28±21.47	138.24±21.47	0.32
DBP	88.96±12.92	84.46±12.40	**0.0001***	89.76±14.38	88.27±11.56	0.61
MBP	112.22±14.61	103.78±12.82	**<0.0001***	111.53±16.65	110.94±12.62	0.87
PP	48.64±13.62	42.14±11.86	**<0.0001***	47.43±13.96	49.68±13.27	0.12
PPI	0.35±0.07	0.33±0.07	**0.0001***	0.34±0.07	0.36±0.07	0.13
HR (bpm)	86.74±14.64	88.11±13.59	0.46	85.88±13.83	87.50±15.32	0.37
RPP (mmHg bpm)	119.93±28.03	111.47±21.65	0.0001*	118.56±29.59	121.12±26.65	0.31
Art stiffness						
AP (mmHg)	10.72±7.01	8.43±5.20	**<0.0001*0.0332***	8.98±6.18	12.23±7.34	**<0.0001*0.0005***
Ref (%)	65.99±7.20	64.83±6.10	0.19	64.48±6.91	67.30±7.21	**<0.0001***0.35
AIx@75 (%)	32.79±11.11	31.55±11.36	**0.0001***	29.20±10.22	35.91±10.95	**0.0022***
PWV (m/s)	7.55±1.08	7.16±1.19	**0.0045***	7.62±1.11	7.49±1.06	**0.0099***
TAS (ml/mmHg)	0.80±0.23	0.76±0.20	0.15	0.77±0.22	6.84±0.22	
PPA	1.33±0.14	1.31±0.14		1.31±0.13	1.31±0.13	
c BP (mmHg)						
cSBP	127.74±17.38	118.28±14.75	**<0.0001***	126.97±19.39	128.42±15.45	**0.27**
cDBP	90.72±13.33	85.93±12.39	**<0.0001***	91.68±14.86	89.90±11.83	0.53
cPP	37.03±11.55	32.5±9.95	**<0.0001***	35.28±11.02	38.56±11.83	**0.0255***
cPP ≥40, no (%)	86 (33%)	39 (15%)	**<0.0001***	32 (27%)	54 (39%)	**0.0354***
Central hemodynamics	5.21±0.86	4.89±0.64	**<0.0001***	5.27±0.99	5.15±0.74	0.82
CO (l/min)	1.29±0.16	1.28±0.15	0.95	1.28±0.17	1.30±0.15	0.11
PR (mmHg/ml)	3.14±0.59	2.91±0.44	**<0.0001***	3.12±0.61	3.17±0.56	0.29
CI (l/min/m^2)	61.25±12.49	56.64±10.48	**<0.0001***	62.15±10.90	60.46±13.72	0.06
SV (ml/beat)	36.89±8.64	33.73±6.59	**<0.0001***	36.52±6.88	37.21±9.93	0.98
SVI (ml/m^2/beat)	123.25±35.92	104.41±28.11	**<0.0001***	124.55±36.57	121.40±33.97	0.47
SW (g m/beat)						

BMI=Body mass index, PA=physical activity, BSA=body surface area, BPC=blood pressure control, ACEI=angiotensin converting enzyme inhibitor, BB=beta blocker, CCB=calcium channel blocker, ARB=angiotensin II receptor blocker, bBP=brachial blood pressure, SBP=systolic blood pressure, DBP=diastolic blood pressure, MBP=mean blood pressure, PP=pulse pressure, PPI=pulse pressure index, HR=heart rate RPP=rate pressure product, AP=augmentation pressure, Ref=reflection percentage, AIx@75=augmentation index at heart rate 75 beats/min, PWV=pulse wave velocity, TAS=total arterial stiffness, PPA=pulse pressure amplification, cSBP=central systolic blood pressure, cDBP=central diastolic blood pressure, cPP=central pulse pressure, CO=cardiac output, PR=peripheral resistance, CI=cardiac index, SV=stroke volume, SVI=stroke volume index, SW=stroke work. **P* less than 0.05 statistically significant

and corrected by antihypertensives) were parameters showing the small insignificant difference. Central hemodynamics were no different between males and females. So, for these parameters, at least gender did not prove to be the significant factor affecting. The mean age was 46 and the female disadvantage can be explained by the peri- and postmenopausal age in most of the females. Apart from gender-specific and sex-hormone-specific differences, results can be viewed in light of shorter height, lesser physical activity, longer mean duration, and lesser use of statins and aspirin compared to males.[20] Stiffness, but not the hemodynamic parameters, showed statistical significance between females and males and suggests the importance of adding this parameter for better understanding of cardiovascular aging. Higher stiffness leads to extra afterload that can explain a higher risk of myocardial infarction in postmenopausal females.

Table 2: Comparison of baseline and study parameters between subgroups of cases based on physical activity (present or absent) and BMI (cutoff 23)

Parameter (unit)	PA+ (*n*=51)	PA (*n*=51)	*P*	BMI <23 (*n*=83)	BMI ≥23 (*n*=83)	*P*
Age (years)	47.12±7.48	47.27±7.45	0.93	50.05±8.05	50.00±7.86	0.99
Male, no (%)	36 (71%)	36 (71%)	1.00	36 (43%)	36 (43%)	1.00
Height (cm)	161.71±5.23	161.67±5.67	0.97	159.18±6.50	160.67±5.74	0.14
Weight (kg)	63.61±9.46	63.90±10.34	0.88	52.78±5.67	66.83±9.27	**<0.0001***
BMI (kg/m^2)	24.20±2.95	24.33±3.06	0.92	20.77±1.57	25.83±2.76	**<0.0001***
BSA (m^2)	1.69±0.15	1.69±0.16	0.99	1.53±0.12	1.72±0.13	**<0.0001***
P A, no (%)	51 (100%)	0 (0%)	–	16 (19%)	12 (14%)	0.53
Duration (years)	4.05±3.08	4.29±3.96	0.81	4.71±3.35	5.11±4.07	0.71
BPC, no (%)	23 (45%)	22 (43%)	1.00	35 (42%)	25 (30%)	0.15
Drugs use						
ACEI, no (%)	38 (76%)	31 (61%)	0.20	52 (63%)	57 (69%)	0.51
BB, no (%)	16 (31%)	23 (45%)	0.22	40 (48%)	49 (59%)	0.21
CCB, no (%)	19 (37%)	24 (47%)	0.42	32 (39%)	31 (37%)	1.00
Diuretics, no (%)	8 (16%)	6 (12%)	0.77	5 (6%)	19 (23%)	**0.0034***
ARB, no (%)	7 (14%)	10 (20%)	0.57	13 (16%)	12 (14%)	1.00
Aspirin, no (%)	14 (28%)	15 (30%)	1.00	16 (19%)	29 (35%)	**0.0355***
Statin, no (%)	14 (28%)	16 (32%)	0.83	12 (14%)	23 (28%)	0.06
bBP (mmHg)						
SBP	134.96±19.30	135.04±16.05	0.72	136.53±22.42	139.41±15.16	**0.34**
DBP	88.51±13.10	88.55±12.39	0.99	88.05±13.17	88.93±11.12	0.64
MBP	109.76±14.94	109.92±13.42	0.59	110.24±16.35	112.07±11.57	0.41
PP	46.45±13.14	46.29±10.11	0.95	48.48±15.18	50.36±12.86	0.18
PPI	0.34±0.07	0.34±0.06	0.90	0.35±0.07	0.36±0.07	0.43
HR (bpm)	88.33±13.90	88.82±14.80	0.78	86.30±15.05	85.95±14.06	0.88
RPP (mmHg bpm)	118.86±26.77	124.05±28.08	0.07	118.56±30.99	119.64±22.52	0.80
Art stiffness						
AP (mmHg)	9.67±5.35	8.96±5.50	0.43	10.36±7.33	12.17±7.13	**0.0492***
Ref (%)	64.57±6.24	65.61±7.15	0.44	66.07±7.28	67.47±7.09	0.17
AIx@75 (%)	31.59±10.99	30.22±10.18	0.61	33.07±11.43	34.39±10.34	0.45
PWV (m/s)	7.29±0.96	7.39±1.03	0.35	7.70±1.30	7.73±0.93	0.90
TAS (ml/mmHg)	0.76±0.22	0.76±0.18	0.94	0.81±0.25	0.83±0.21	0.46
PPA	1.33±0.12	1.35±0.15	0.66	1.35±0.15	1.29±0.13	**0.0141***
c BP (mmHg)						
cSBP	125.55±17.53	125.25±15.27	0.93	126.27±19.66	130.24±14.19	0.14
cDBP	90.31±13.52	90.45±13.08	0.96	89.92±13.58	90.81±11.51	0.63
cPP	35.24±10.87	34.78±8.94	0.95	36.35±11.78	39.48±11.79	**0.0441***
cPP ≥40, no (%)	16 (32%)	9 (18%)	0.17	22 (27%)	40 (48%)	**0.0062***
Central hemodynamics	5.16±0.87	5.17±0.77	0.64	5.16±0.96	5.38±0.38	0.64
CO (l/min)	1.29±0.13	1.28±0.13	0.83	1.29±0.17	1.26±0.14	0.39
PR (mmHg/ml)	3.08±0.49	3.09±0.50	0.85	3.41±0.63	3.13±0.47	**<0.0001***
CI (l/min/m^2)	60.14±8.72	61.71±10.64	0.57	61.34±15.85	59.52±9.27	0.49
SV (ml/beat)	35.73±5.64	36.74±7.03	0.43	40.45±11.00	36.00±7.06	0.0016*
SVI (ml/m^2/beat)	118.11±30.11	120.74±28.41	0.65	122.18±42.66	124.86±30.95	0.16
SW (g m/beat)						

PA+ = Physical activity present, PA=physical activity absent; rest of the abbreviations are the same as Table 1. **P* less than 0.05 statistically significant

Physical activity had no significant impact on PWA results in contrast to Guimaraes *et al.*[21]. It can be due to the moderate intensity of exercise judged by self-reporting. Weight loss and exercise are more significant in obese and type 2 diabetics,[22] which was not the case in our study with nondiabetic individuals with mean BMI 24. Using BMI cutoff 23, we found BMI < 23 to be advantageous for few of PWA parameters. BMI as a confounder for cardiovascular aging is known and documented by the previous study like ours.[23] However, visceral obesity can give better impact than BMI-guided general obesity, in line with our previous study showing the fact that fat quality is more important than quantity in our population.[24] Despite no evident significance, physical activity and goal of BMI < 23 both are good practices for overall well-being.

Blood pressure control had an advantage for few parameters we studied in the case group. As such, most parameters are dependent on blood pressure but with a varying grade. In general, controlled blood pressure was absent in majority and it denies

Table 3: Comparison of baseline and study parameters between subgroups of cases based on blood pressure control (present or absent) and duration (cutoff 5)

Parameter (unit)	BPC+ (*n*=95)	BPC− (*n*=95)	*P*	Duration <5 (*n*=151)	Duration ≥5 (*n*=107)	*P*
Age (years)	48.86±6.31	48.67±6.30	0.97	48.16±8.09	49.42±6.82	0.16
Male, no (%)	47 (49%)	47 (49%)	1.00	77 (51%)	64 (60%)	0.17
Height (cm)	160.88±5.38	160.79±6.03	0.64	160.70±5.90	160.43±5.53	0.29
Weight (kg)	63.45±10.81	63.05±10.00	0.84	63.54±10.80	62.93±10.14	0.86
BMI (kg/m^2)	24.44±3.61	24.34±3.39	0.85	24.53±3.56	24.40±3.31	>0.99
BSA (m)	1.68±0.16	1.68±0.16	0.76	1.68±0.17	1.67±0.16	0.87
P A, no (%)	23 (24%)	13 (14%)	0.09	35 (23%)	16 (15%)	0.12
Duration (years)	4.63±3.31	5.46±3.89	0.19	2.35±1.05	8.44±3.25	**<0.0001***
BPC, no (%)	95 (100%)	0 (0%)	–	61 (40%)	37 (35%)	0.36
Drugs use						
ACEI, no (%)	62 (65%)	63 (66%)	1.00	102 (68%)	71 (66%)	0.89
BB, no (%)	48 (51%)	49 (52%)	1.00	74 (49%)	54 (50%)	0.90
CCB, no (%)	45 (47%)	34 (36%)	0.14	49 (32%)	48 (45%)	0.0505
Diuretics, no (%)	12 (14%)	15 (17%)	0.68	20 (13%)	17 (16%)	0.18
ARB, no (%)	19 (21%)	16 (18%)	0.71	30 (20%)	14 (13%)	0.59
Aspirin, no (%)	32 (34%)	35 (37%)	0.76	44 (29%)	32 (30%)	0.89
Statin, no (%)	23 (25%)	28 (30%)	0.51	37 (25%)	23 (21%)	0.65
bBP (mmHg)						
SBP	121.52±9.92	147.12±16.11	**<0.0001***	137.62±18.36	138.03±20.27	0.85
DBP	78.57±8.09	94.31±11.27	**<0.0001***	89.66±12.91	87.97±12.95	0.22
MBP	98.13±8.08	110.33±11.65	**<0.0001***	111.63±14.31	110.64±15.08	0.68
PP	43.05±8.00	52.18±15.36	**<0.0001***	47.96±12.84	49.59±14.65	0.31
PPI	0.35±0.05	0.35±0.08	0.58	0.35±0.07	0.36±0.07	0.26
HR (bpm)	84.68±15.29	87.22±11.80	0.15	86.47±14.06	87.13±15.48	0.72
RPP (mmHg bpm)	103.10±21.26	128.37±23.22	**<0.0001***	119.47±27.38	120.58±29.05	0.88
Art stiffness						
AP (mmHg)	9.55±4.98	11.52±8.31	0.20	10.47±6.81	11.07±7.28	0.43
Ref. (%)	67.14±6.21	65.43±7.30	0.08	66.26±7.16	65.61±7.27	0.72
AIx@75 (%)	32.09±11.10	32.53±11.02	0.49	32.34±10.78	33.73±11.22	0.24
PWV (m/s)	7.00±0.78	7.84±0.95	**<0.0001***	7.51±1.15	7.61±0.98	0.47
TAS (ml/mmHg)	0.78±0.18	0.81±0.26	0.32	0.80±0.24	0.81±0.23	0.31
PPA	1.31±0.12	1.34±0.15	0.08	1.32±0.13	1.34±0.15	0.19
c BP (mmHg)						
cSBP	113.17±10.42	135.81±13.95	**<0.0001***	128.29±17.19	126.97±17.70	0.67
cDBP	79.77±8.29	96.36±11.43	**<0.0001***	91.48±13.24	89.65±13.44	0.24
cPP	33.39±7.63	39.51±13.08	**0.0013***	36.83±11.49	37.32±11.69	0.58
cPP ≥40, no (%)	19 (20%)	39 (41%)	**0.0026***	47 (31%)	39 (36%)	0.42
Central hemodynamics	4.69±0.51	5.51±0.86	**<0.0001***	5.20±0.84	5.22±0.90	0.98
CO (l/min)	1.26±0.12	1.31±0.20	0.68	1.29±0.15	1.29±0.18	0.50
PR (mmHg/ml)	2.81±0.41	3.32±0.56	**<0.0001***	3.14±0.58	3.14±0.59	0.98
CI (l/min/m^2)	56.89±10.93	63.55±8.50	**<0.0001***	61.40±13.25	61.04±13.39	0.98
SV (ml/beat)	34.24±7.71	38.16±6.13	**<0.0001***	39.40±9.26	36.81±7.72	0.93
SVI (ml/m^2/beat)	99.88±22.05	135.71±30.64	**<0.0001***	122.84±34.92	122.89±35.69	0.95
SW (g m/beat)						

BPC+ = Blood pressure control present, BPC− = blood pressure control absent; rest of the abbreviations are the same as Table 1. **P* less than 0.05 statistically significant

complete reversal or halting of accelerated cardiovascular aging in hypertension. Controlled blood pressure had a significant effect mainly on central hemodynamics and lesser effect on arterial stiffness parameters. This indicates that later parameters are superior to give brachial blood pressure-independent inference.[25] We did not find a significant difference of duration less than or more than 5 years with respect to study results, in line with the previous study.[3] It indicates the importance of disease, its early diagnosis, and prompt treatment more than the chronicity of this incurable disease.

By multiple linear regressions, we studied predictors of major study parameters. Except PWV, age, height, weight, and BMI were not significant predictors of study outcome parameters. Similarly, most of these were independent of brachial systolic, diastolic, and mean blood pressures pointing toward the utility of these parameters to complement routine blood pressure measurement. Heart rate and pulse pressure proved to be the most consistent predictors that one can infer from pulse examination and proved to be the potential of details one can obtain by the arterial pulse. Age was a very strong predictor of aPWV in line with available

Table 4: Calculation of predictors for dependent variables by multiple linear regressions ($r_{partial}$ values) in the case group

	AP	AIx@75	aPWV	TAS	cSBP	cDBP	cPP	CO	SW
Age	0.01	0.00	**0.84****	−0.04	0.01	0.05	−0.04	0.02	−0.07
Height	−0.03	−0.04	0.05	0.03	−0.06	0.00	0.05	−0.05	−0.04
Weight	0.01	0.01	−0.04	−0.04	0.06	0.01	−0.03	0.06	0.05
BMI	−0.01	−0.01	0.03	0.03	−0.04	−0.03	0.03	−0.06	−0.05
SBP	0.01	0.05	0.00	0.01	0.04	−0.04	0.02	0.01	**0.14***
DBP	0.03	0.02	0.00	0.06	0.08	0.03	0.03	−0.06	−0.02
MBP	−0.05	−0.04	0.07	−0.10	**0.14***	**0.72****	−0.01	0.10	0.06
HR	**−0.33****	**0.45****	0.04	**0.65****	**−0.17***	**0.37****	**−0.20***	**0.42****	**−0.67****
PP	**0.17***	0.07	0.09	**0.28****	**0.25****	**−0.68****	0.05	−0.01	0.03
Duration	−0.03	0.01	−0.04	−0.04	**−0.15***	−0.08	0.05	0.01	−0.01

Abbreviations are the same as Table 1. **P* less than 0.05 statistically significant, ***P* less than 0.0001 extremely statistically significant

literature.[26] We found central blood pressures to be not predicted significantly by corresponding brachial artery values. This can be due to (1) elastic aorta versus muscular brachial artery, (2) proximity of aorta than other arteries to heart, (3) different effect of vascular aging pathology on arteries, and (4) different impact of antihypertensives in central and peripheral arteries. Thus, central blood pressures are adding new information on existing brachial blood pressure and should be used optimally.[3,5,27]

Hypertension is a growing epidemic with serious aftermaths on cardiovascular and overall health. PWA offers a better understanding of the same, and with the availability of devices like Mobil-O-graph, it has the potential to be applicable and studied further. In our two studies,[9,28] we found that PWA parameters were significantly higher in apparently healthy, normotensive, young, nonobese offspring of the diabetic or hypertensive parent. This suggests that PWA parameters like arterial stiffness can help in early diagnosis and a better understanding of disease course. In another study done in our population, we have shown that diabetics not using any antihypertensive had an adverse profile of beyond brachial blood pressure discrete cardiovascular parameters, independent of the duration and glycemic control, related to gender, BMI, and physical activity, indicating vascular progeria in the absence of hypertension.[3] So, diabetes or hypertension alone can be studied better by PWA parameters, inferring to central hemodynamics and arterial stiffness. But these, being a baseline study, call for further interventional or vertical studies for consolidation of our results.

There were few limitations of our study like cross-sectional nature, moderate sample size, lack of baseline data or follow-up, and absence of biochemical investigations. Use of novel instrument Mobil-O-graph can be considered more a strength than limitation of the study.

Conclusion

Oscillometric PWA revealed the adverse profile of arterial stiffness and central hemodynamics in treated Gujarati hypertensives, associated with female gender, BMI, and blood pressure control, predicted mainly by heart rate and pulse pressure. These potential and beyond brachial blood pressure parameters allow assessment of discrete and direct cardiovascular functioning that needs to be confirmed further.

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Authors' contribution

- JDS contributed in the conception of the work, conducting the study, acquisition of data, analysis and interpretation of data for the work, drafting of the manuscript, approval of the final version of the manuscript, and agreed for all aspects of the work
- HBM, HiBM, and SJP contributed in conducting the study and revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work
- CJS contributed in revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work.

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Conflicts of interest

There are no conflicts of interest.

References

1. Mohan P, Mohan SB, Dutta M. Communicable or noncommunicable diseases? Building strong primary health care systems to address double burden of disease in India. J Family Med Prim Care 2019;8:326-9.
2. Yu S, Chi C, Protogerou AD, Safar ME, Blacher J, Argyris AA, *et al.* 24-hour aortic blood pressure variability showed a stronger association with carotid damage than 24-hour

brachial blood pressure variability: The SAFAR study. J Clin Hypertens 2018;20:499-507.

3. Solanki JD, Munshi HB, Mehta HB, Shah CJ. Central hemodynamics and arterial stiffness in Gujarati diabetics not receiving any antihypertensive: A case–control study based on oscillometric pulse wave analysis. J Family Med Prim Care 2019; Ahead of Print.
4. Milan A, Degli Esposti D, Salvetti M, Izzo R, Moreo A, Pucci G, *et al.* Prevalence of proximal ascending aorta and target organ damage in hypertensive patients: The multicentric ARGO-SIIA project (Aortic RemodellinG in hypertensiOn of the Italian Society of Hypertension). J Hypertens 2019;37:57-64.
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.
7. Weiss W, Gohlisch C, Harsch-Gladisch C, Tölle M, Zidek W, van der Giet M. 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. Hypertens 2011;58:825-32.
9. Solanki JD, Mehta HB, Shah CJ. Pulse wave analysed 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. Oscillometric pulse wave analysis in newly diagnosed never treated Gujarati hypertensives. Vasc Invest Ther 2018;1:62-7.
11. Xaplanteris P, Vlachopoulos C, Protogerou AD, Aznaouridis K, Terentes-Printzios D, Argyris AA, *et al.* A clinical score for prediction of elevated aortic stiffness: Derivation and validation in 3943 hypertensive patients. J Hypertens 2019;37:339-46.
12. Yeboah K, Antwi DA, Gyan B. Arterial stiffness in nonhypertensive type 2 diabetes patients in Ghana. Int J Endocrinol 2016;2016:6107572.
13. Solanki JD, Makwana AH, Mehta HB, Gokhale PA, Shah CJ. Hypertension in type 2 diabetes mellitus: Effect of the disease and treatment on development of peripheral artery disease. Int J Diabetes Dev Ctries 2015;35(Suppl 3):S380-4.
14. Solanki JD, Basida SD, Mehta HB, Panjwani SJ, Gadhavi BP. Comparative study of cardiac autonomic status by heart rate variability between under-treatment normotensive and hypertensive known type 2 diabetics. Indian Heart J 2017;69:52-6.
15. Solanki JD, Mehta HB, Panjwani SJ, Munshi HB, Shah CJ. Impact of concomitant use of beta blocker, statin, aspirin, and metformin on central hemodynamics and arterial stiffness in hypertension. J Pharmacol Pharmacother 2018;9:167-73.
16. Townsend RR, Black HR, Chirinos JA, Feig PU, Ferdinand KC, Germain M, *et al.* Clinical use of pulse wave analysis: Proceedings from a symposium sponsored by North American Artery. J Clin Hypertens 2015;17:503-13.
17. Li Y, Hickson SS, McEniery CM, Wilkinson IB, Khir AW. Stiffening and ventricular–arterial interaction in the ascending aorta using MRI: Ageing effects in healthy humans. J Hypertens 2019;37:347-55.
18. Aparicio LS, Barochiner J, Peuchot VA, Giunta DH, Martínez R, Morales MS, *et al.* Comparing office, central, home and ambulatory blood pressure in predicting left ventricular mass. Hipertens Riesgo Vasc 2019;36:5-13.
19. Chung TH, Shim JY, Kwon YJ, Lee YJ. High triglyceride to high-density lipoprotein cholesterol ratio and arterial stiffness in postmenopausal Korean women. J Clin Hypertens 2019;21:399-404.
20. 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.
21. Guimaraes GV, Ciolac EG, Carvalho VO, D'Avila VM, Bortolotto LA, Bocchi EA. Effects of continuous vs. interval exercise training on blood pressure and arterial stiffness in treated hypertension. Hypertens Res 2010;33:627.
22. Urbina EM, Kimball TR, Khoury PR, Daniels SR, Dolan LM. Increased arterial stiffness is found in adolescents with obesity or obesity-related type 2 diabetes mellitus. J Hypertens 2010;28:1692.
23. Brunner EJ, Shipley MJ, Ahmadi-Abhari S, Tabak AG, McEniery CM, Wilkinson IB, *et al.* Adiposity, obesity, and arterial aging: Longitudinal study of aortic stiffness in the Whitehall II cohort. Hypertens 2015;66:294-300.
24. Solanki JD, Makwana AH, Mehta HB, Gokhale PA, Shah CJ. Body composition in type 2 diabetes: Change in quality and not just quantity that matters. Int J Prev Med 2015;6:122.
25. Safar ME. Arterial stiffness as a risk factor for clinical hypertension. Nat Rev Cardiol 2018;15:97-105
26. Solanki JD, Mehta HB, Shah CJ. Aortic pulse wave velocity and augmentation index@75 measured by oscillometric pulse wave analysis in Gujarati nonhypertensives. Vasc Invest Ther 2018;1:50-5.
27. Yannoutsos A, Rinaldi ER, Zhang Y, Protogerou AD, Safar ME, Blacher J. Central hemodynamics in risk assessment strategies: Additive value over and above brachial blood pressure. Curr Pharm Des 2015;21:719-29.
28. 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;23:72.