

Vascular Changes in Hypertension

Dr.K.Muralidharan M.D. , Dr.Sanjay.C M.B.B.S

Assistant Professor Of General Medicine

Madha Medical College And Research Institute Affiliated To Tamil Nadu Dr.M.G.R.Medical University

Corresponding auther: Dr.K.Muralidharan M.D

Abstract: Increasing evidence shows arterial hypertension produces early vascular changes at the microvascular and macrovascular levels;also vascular changes in increasing age predisposes to arterial stiffness. **METHODS:** In a cohort of 121 adults subjects we have done pulse wave analysis (PWA) measurement, blood pressure (BP), Hemodynamic changes, Arterial stiffness, Augmentation index (AIx) using Mobil-O-Graph. We measured among normotensive (NT) , hypertensive (HT) adults. AIx was derived by PWA using Mobil-O-Graph. **RESULTS:** AIx increasing with increase in risk scores and increases significantly with cardiovascular risk factors. Pulse wave velocity increases with risk scores. With high BMI the central systolic blood pressure (cSBP), central diastolic blood pressure (cDBP), central pulse pressure (cPP) increases in HT group. Vascular age (VA) increases with biological age and also due to poor life style and other comorbidities. **CONCLUSION:** Comparing and measuring the above mentioned parameters in HT adults with control group increased arterial stiffness with biological ageing, arteriosclerosis associated with increased cardiovascular adverse events. Thus maintaining the health of the vessel is very important to control end organ damage and very critical in overall BP management.

Date of Submission: 09-05-2018

Date of acceptance: 26-05-2018

I. Introduction:

Morbidity and mortality in hypertension are related primarily to structural and functional alterations of arterial wall [1-3]. Changes in the arterial wall can lead to increased arterial stiffness which has been shown to influence cardiovascular prognosis adversely [4]. Thus measuring arterial stiffness has been recommended in the preventative measurement of cardiovascular disease [5]. To examine arterial elastic properties PWA is used increasingly in clinical studies as it is simple and non-invasive technique. The PWA measures AIx, the parameter that reflects the degree to which cAP is enhanced by the wave reflection of the pulse wave. AIx is a useful index of cardiovascular risk but an association between AIx and cardiovascular risk as not yet been confirmed. In the normal arterial system there is a steep gradient of increasing arterial stiffness moving outward from the heart. This pulse wave velocity (PWV) closely correlate of arterial wall stiffness is only 4-6m/sec in the highly compliant proximal aorta and increases 8-10 m/sec in the stiffer peripheral muscular arteries. This progressive increase in regional arterial stiffness together with branching and narrowing of the lumen, creates an impedance mismatch and leads to partial reflection of the advancing pressure wave. Prior studies have shown that central arterial stiffness increases to a far greater extent than peripheral arterial stiffness with advancing age. As a result aortic stiffness may equal or exceed peripheral arterial stiffness in the elderly. The number of non pathological changes that influence AIx includes age, blood pressure, heart rate and height have been described (6,7,8).

II. Methods:

In this cross sectional study 81 adults (HT) of varying age group in both sexes who are enrolled from our outpatient clinic based on the medical history and clinical investigations. Additionally 40 healthy adults (NT) were investigated.

Table 1 :PATIENTS CHARACTERISTICS AND ASSESSMENT OF RISK FACTORS:

VARIABLE	MEN	WOMEN
Age, years	71	50
Height	184	140
Weight	67	52
Brachial Systolic Pressure, mmHg	149	116
Brachial Diastolic Pressure, mmHg	98	73
Mean Arterial Pressure, mmHg	122	92
Brachial Pulse Pressure, mmHg	51	43
Heart Rate, bpm	85	78
Body Mass Index, kg/sq.m	24.3	22.4
Ratio of Total/HDL Cholesterol	4.2	3.2
Triglycerides, mg/dL	194	215
Fasting Glucose, mg/dL	122	13

III. Blood Pressure:

Participants were seated and rested for 5 minutes in a quiet environment after which their BP was measured sphygmomanometrically 3 times, at 5-minute interval, with the auscultatory method over the brachial artery. The mean between these measurements was used for further analysis. Participants were considered to be NT if both SBP and DBP or both that was above 90th percentile but below the 95th percentile, or if their BP exceeded 120/80 mmHg even if either BP component was below the 90th percentile. Hypertension was defined as a systolic or a diastolic BP or both that was the 95th percentile.

Mobil-O-Graph



MOBIL-O-GRAPH: The Pulse wave analysis monitor provides information about vital hemodynamic parameters connected with the stresses of everyday life

ARTERIAL STIFFNESS: The PWA monitor enables validated assessment of arterial stiffness.

HEMODYNAMICS: An aid to clinical decisions hemodynamic monitoring enables for the first time a better insight into a patient's pathophysiology and provides clinical support for diagnosis and treatment. PWA is displayed in tables and easy to follow graphical reports, providing a comprehensive statistical assessment.

METHOD: Oscillometric

Parameters Of Mobil-O-Graph	Normal Range
Central systolic Pressure	Depends on age
Central Diastolic Pressure	60 to 80 mmHg
Stroke Volume	70ml
Cardiac Output	4 to 5 L/min
Total vascular resistance	5 to 1.2 mm/Hg/ml

Cardiac Index	2.5 to 4.2L/min/m.sq
Body surface area	1.9 m.sq in men and 1.6 m.sq in women
PULSE WAVE VELOCITY	
AGE(Years)	MEAN PWV(m/s)
10-19	5.04
20-29	5.86
30-39	6.32
40-49	6.85
50-59	8.15
60-69	8.47
>70	9.01

Table 2: Basal variables of measured variables in study population

	TOTAL	NT	HT
Age, years	121	40	81
BMI, kg/sq.m	23.2	22.5	28.7
Body Height, m	162	167	155
Body Weight, kg	59.5	58.6	61.3
Peripheral SBP, mmHg	133	126	149
Peripheral DBP, mmHg	86	75	94
Peripheral PP, mmHg	47	42	51
Central SBP, mmHg	121	118	131
Central DBP, mmHg	88	80	92
AIx, %	26	24	28.4
PWV	8.9	8.15	8.47

*Values are given as mean +/- SD*The difference between NT and HT is significant at P<0.05 Abbreviations: AIx, Augmentation Index ; BMI, Body Mass Index ; SBP, Systolic Blood Pressure ; DBP, Diastolic Blood Pressure ; PP, Pulse Pressure ;PWV, Pulse Wave Velocity

IV. Discussion:

Our study involving 81 HT patient and 40 NT patient investigated micro and macrovascular parameters in relation to BP at baseline. We found that AIx was correlated with central SBP and DBP, no significant correlation between AIx and peripheral SBP or DBP and no difference in AIx among the three BP categories in our study, presumably because of the shorter cumulative exposure to higher BP in adolescents as compared with adults. Because changes in the elasticity of the arterial walls and in pulse-wave reflection patterns caused by long-standing hypertension can play a role in the pathogenesis of cardiovascular disease, pulse-wave analysis has become clinically useful(9,10,11,12). Furthermore, a very recent, large study showed that brachial-ankle pulse-wave velocity (PWV), a marker of arterial stiffness, is an independent predictor of longitudinal increases in BP and new-onset HT(13). Central SBP, central AoPP, and AIx are determined by arterial stiffness, the amplitude of reflected pulse waves, the point of pulse-wave reflection, and the duration and pattern of ventricular ejection(14). Accordingly, AIx is more than a marker of arterial stiffness, reflecting rather the overall interaction between the arterial tree and the left ventricle(14). In both NT (15) and HT adults,(16) a higher AIx is associated with increased left-ventricular mass. In a large cohort of healthy, NT individuals, the AIx of participants under 20 years of age was $-2 \pm 8\%$ in men and $5 \pm 10\%$ in women(17). AIx values were higher overall in women than in men and for each decade of life(17) We did not find any significant gender difference in AIx in our adolescent study population.

In patients with atherosclerotic disease, augmentation index was significantly correlated with SBP, DBP, HR and height, determined by monivariate linear regression analysis. In contrast, augmentation index was not correlated to age in patients with atherosclerotic disease. Furthermore, augmentation index was not correlated with the presence of vasoactive medication in subjects with atherosclerotic disease. After multiple regression analysis, augmentation index remained correlated to DBP, HR, height, and gender but no longer to SBP.

Figure.1.Relationship between Central SBP and Augmentation Index of Normotensives

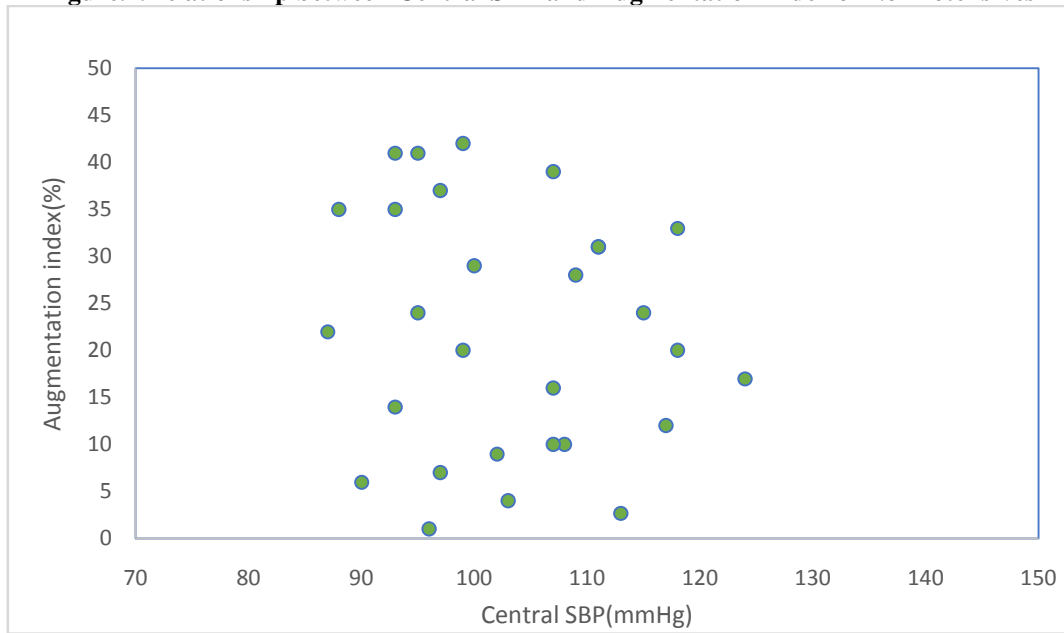
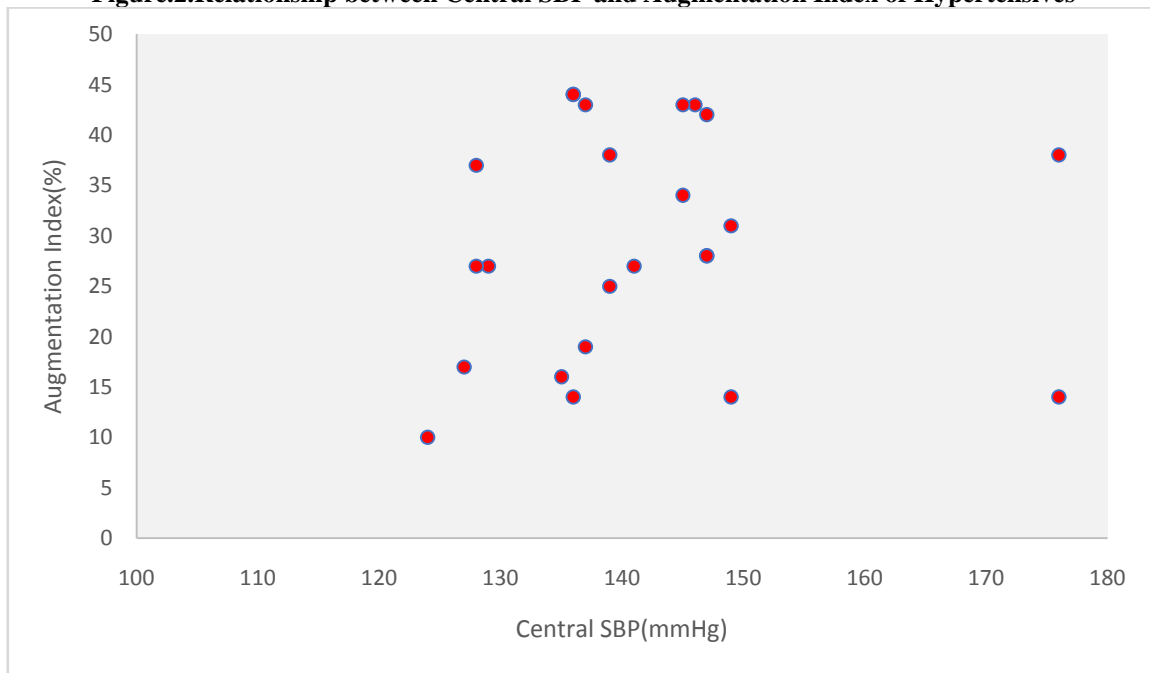


Figure.2.Relationship between Central SBP and Augmentation Index of Hypertensives



V. Risk Scores:

Using the ‘coronary risk chart’ of the European Society of Cardiology (ESC risk score) the absolute risk (%) of developing coronary heart disease (non-fatal coronary heart disease or coronary death) over the next 10 years was estimated for individuals who had no evidence of clinically symptomatic coronary heart or atherosclerotic disease [18]. This algorithm applies a risk function derived from the Framingham study which is based on the following risk factors: gender, age, smoking status, systolic blood pressure, total cholesterol and presence of diabetes mellitus [19]. Patients were then categorized into five risk levels (low: under 5%; mild: 5–10%; moderate: 10–20%; high: 20–40%; very high: over 40% risk of developing coronary heart disease over the next 10 years). Using the ESC risk score, the 144 patients without a previous history of coronary heart or atherosclerotic disease were classified into five risk groups (low, mild, moderate, high risk, very high risk). Augmentation index increased from the lowest to the highest risk class, with a significant difference between risk classes determined by Kruskal–Wallis ANOVA. Augmentation index was strongly correlated with ESC risk score. Applying the SMART risk score, all subjects were classified into risk levels. Risk levels ranged from

0 to 15 points, with a mean of 6.4 points Augmentation index rose significantly with increasing cardiovascular risk determined by SMART risk score. Augmentation index was strongly correlated with the SMART risk score.

Table 3: Basal values of measured variables in gender .population

	MALE	FEMALE
Age, years	57	56
BMI, kg/sq.m	24.3	22.4
Peripheral SBP, mmHg	149	116
Peripheral DBP, mmHg	98	73
Peripheral PP	51	43
Central SBP, mmHg	128	114
Central DBP, mmHg	95	80
Aix, %	24	27.8
PWV	9.1	8.6

*Values are given as mean (+/- SD)

*The difference between male and female is significant at P<0.05

Abbreviations :Aix, Augmentation Index ; BMI, Body Mass Index ; SBP, Systolic Blood Pressure ; DBP, Diastolic Blood Pressure ; PP, Pulse Pressure ; PWV, Pulse Wave Velocity

TABLE 4: Results for dependent variables in analysis of regression against Central systolic blood pressure

	BETA	STANDARD DEVIATION	P-Value
BMI,kg/m.sq	0.8188	1.3435	<0.05
Aix,%	0.9155	2.6870	<0.05

VI. Conclusion:

It has been reported recently, in a study involving British subjects, that cholesterol may determine augmentation index in healthy subjects [20]. In the present study, this association did not reach statistical significance by multiple regression analysis. It is possible that this lack of a correlation may be due to dietary or genetic differences. In summary, our cross-sectional study supports an association between augmentation index and the risk of cardiovascular and total mortality. Although we can not conclude that augmentation index predicts cardiovascular risk, our results support the use of augmentation index in clinical studies in which arterial stiffness is assessed. Longitudinal studies are necessary to determine whether augmentation index predicts mortality, thus justifying the measurement of arterial function in preventative medicine. In addition, augmentation index was correlated to age only in healthy subjects, and thus corrections for age should be applied with caution when augmentation index is used in studies involving patients who have cardiovascular disease.

Reference:

- [1]. Asmar R, Benetos A, London G, Hugue C, Weiss Y, Topouchian J, et al. Aortic distensibility in normotensive, untreated and treated hypertensive patients. *Blood Press* 1995; 4:48–54.
- [2]. O'Rourke M. Mechanical principles in arterial disease. *Hypertension* 1995; 26:2–9.
- [3]. Nichols WW, O'Rourke MF. McDonald's blood flow in arteries. Theoretical, experimental and clinical principles. 4th ed. London: Edward Arnold; 1998.
- [4]. O'Rourke MF, Mancia G. Arterial stiffness. *J Hypertens* 1999; 17:1–4.
- [5]. Guidelines Subcommittee. 1999 World Health Organization–International Society of Hypertension Guidelines for the Management of Hypertension. *J Hypertens* 1999;
- [6]. Kelly R, Hayward C, Avolio A, O'Rourke M. Noninvasive determination of age-related changes in the human arterial pulse. *Circulation* 1989;
- [7]. Hayward CS, Kelly RP. Gender-related differences in the central arterial pressure waveform. *J Am Coll Cardiol* 1997; 30:1863–1871.
- [8]. McGrath B, Liang YL, Kotsopoulos D, Cameron J. Impact of physical and physiological factors on arterial function. *Clin Exp Pharmacol Physiol* 2001;
- [9]. Nichols W, O'Rourke M. McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles. Arnold, London, 2005.
- [10]. O'Rourke M. Mechanical principles in arterial disease. *Hypertension* 1995; 26:2–9.
- [11]. Numburger J, Keflioglu-Scheiber A, Opazo Saez AM, Wenzel RR, Philipp T, Schafers RF. Augmentation index is associated with cardiovascular risk. *J Hypertens* 2002; 20:2407–2414.
- [12]. Covic A, Haydar AA, Bhamra-Ariza P, Gusbeth-Tatomir P, Goldsmith D. Aortic pulse wave velocity and arterial wave reflections predict the extent and severity of coronary artery disease in chronic kidney disease patients. *J Nephrol* 2005; 18:388–396.

- [13]. Takase H, Dohi Y, Toriyama T, Okado T, Tanaka S, Sonoda H, Sato K, Kimura G. Brachial-ankle pulse wave velocity predicts increase in blood pressure and onset of hypertension. *Am J Hypertens* 2011; 24:667–673.
- [14]. Safar ME, London GM. Therapeutic studies and arterial stiffness in hypertension: recommendations of the European Society of Hypertension. The Clinical Committee of Arterial Structure and Function. Working Group on Vascular Structure and Function of the European Society of Hypertension. *J Hypertens* 2000; 18:1527–1535.
- [15]. Saba PS, Roman MJ, Pini R, Spitzer M, Ganau A, Devereux RB. Relation of arterial pressure waveform to left ventricular and carotid anatomy in normotensive subjects. *J Am Coll Cardiol* 1993; 22:1873–80.
- [16]. Lekakis JP, Zakopoulos NA, Protogerou AD. Cardiac hypertrophy in hypertension: relation to 24-h blood pressure profile and arterial stiffness. *Int J Cardiol* 2004; 97: 29–33.
- [17]. McEniery CM, Yasmin, Hall IR, Qasem A, Wilkinson IB, Cockcroft JR. Normal vascular aging: differential effects on wave reflection and aortic pulse wave velocity. The Anglo-Cardiff Collaborative Trial (ACCT). *J Am Coll Cardiol* 2005; 46:1753–1760.
- [18]. Prevention of coronary heart disease in clinical practice. Recommendations of the Second Joint Task Force of European and other Societies on coronary prevention. *Eur Heart J* 1998; 19:1434–1503.
- [19]. Anderson KM, Wilson PW, Odell PM, Kannel WB. An updated coronary risk profile. A statement for health professionals. *Circulation* 1991; 83:356–362.
- [20]. Wilkinson IB, Prasad K, Hall IR, Thomas A, MacCallum H, Webb DJ, et al. Increased central pulse pressure and augmentation index in subjects with hypercholesterolemia. *J Am Coll Cardiol* 2002; 39:1005–1011.

Dr.K.Muralidharan M.D "Vascular Changes In Hypertension " IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 17, no. 5, 2018, pp 01-06