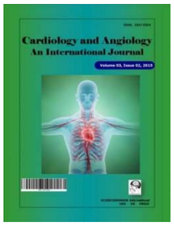

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Carotid-radial Pulse Wave form and Velocity in Normotensive and Hypertensive Pregnant Women at University Teaching Hospital (UTH), Lusaka, Zambia

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Authors' contributions

This work was a collaborative effort from the Cardiovascular Science Laboratory at the University of Zambia School of Medicine. Author LK formulated and implemented the study protocol drafted the manuscript. Author BV was the clinical supervisor of the study protocol. Author MJ was a peer supporter and participated in literature review while author FMG was the CVS Laboratory Director, main academic supervisor and coordinated the script writing process. All authors read and approved the final manuscript.

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ABSTRACT

Background: Hypertensive disease in pregnancy continues to be one of the leading causes of maternal death. Pregnancy induced hypertension (PIH) is said to be accompanied by several cardiovascular pathophysiological changes including increases in arterial stiffness. Pulse wave velocity (PWV) is a method for measuring arterial stiffness. Both the pulse wave form and the velocity are said to change in PIH. However, studies documenting these characteristics of the pulse wave have mainly been in the Caucasian population.
Aims and Objectives: To establish the characteristics of the carotid-radial (cr) pulse wave in

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normotensive (NTN) and hypertensive (HTN) pregnant black African women at the UTH in Lusaka, Zambia.

Methodology: This cross-sectional study comprised of 26 systemically selected pregnant women between the ages 18-45 years old who met the criteria. A structured interview was used to collect socio demographic data. Anthropometric measurements were taken. After a 15 minute rest, peripheral systolic and diastolic blood pressures (BP) were measured. The PWV measurement involved applying non-invasive piezoelectric sensors on the skin over the carotid artery in the neck and the radial artery on the wrist (carotid-radial segment crPWV). Using IBM® SPSS® version 20.0 analyses were made using mann - whitney and spearman correlation tests. A 95% confidence interval (CI) and *P*-value of <0*.05* were set. Quality recordings were obtained from the crPWV recording processes showing the wave forms and specific measurements were made.

Results: The anthropometric measurements were comparable between the 2 groups. There were significant changes in the pulse wave forms. While the normotensive participants had the type C wave form, the type A wave form was recorded from hypertensive participants. The augmentation pressure (AP) in NTN was 4±5 mmHg while it was 9±8 mmHg in HTN, indicating an increase in pressure difference from the systolic shoulder to the peak of the pulse wave ($p < 0.05$). There was also a significant increase in the augmentation index (Aix) (1±22% vs 16±23%) ($p<0.05$). The hypertensive pregnant women also had a significantly higher PWV (9±4 m/s vs 13±7 m/s) ($p<0.05$).

Conclusion: Distinct differences were seen in the cr pulse wave forms and velocity between normotensive and hypertensive individuals with PIH indicating an increase in arterial stiffness. These findings suggest the presence of significant peripheral vascular changes that may underly the pathophysiology of PIH.

Keywords: Pulse wave velocity (PWV); pregnant women; hypertension; augmentation pressure; augmentation index.

1. INTRODUCTION

Pulse wave velocity (PWV), the speed at which the pulse wave travels on an arterial segment, is considered the “gold standard” in the measurement of arterial stiffness [1,2]. It is described as a simple, non-invasive and reproducible method of measurement [2]. It has also been described as a strong predictor for future cardiovascular (CV) events [3].

The pulse wave carries information on how blood is propagated along an arterial segment and not only systolic and diastolic pressures as measured by a sphygmomanometer. Both the pulse wave form and the pulse wave velocity are definitive of vascular health. Determinants of the wave form are ‘ejection pattern of the left ventricle, the mechanical properties of the arterial system, and the peripheral vascular resistance’ [4]. The pulse wave velocity can be measured between the carotid radial arterial system it spans called carotid-radial PWV (crPWV). Carotid-radial PWV is said to be a measure of muscular artery stiffness [5].

Several hemodynamic changes have been reported in pregnancy due to several physiological and/or pathophysiological processes that occur at the different stages of pregnancy. Among the pathophysiological processes reported in pregnancy induced hypertension (PIH) is endothelial dysfunction which causes increased arterial stiffness. This is said to be caused mainly by the release of toxic substances that consequently inhibit the bioavailability of nitric oxide which is a vasodilator [6,7,8,9,10]. Indeed PWV is said to increase significantly in women with PIH when compared to normotensive pregnant controls [11]. However, these findings have been mostly in the Caucasian population. Data in African women is lacking.

Pregnancy induced hypertension (PIH) is very common in the African population. Its prevalence in South Africa was reported as 21.6% in 2012 [12] and at the University Teaching Hospital (UTH) Lusaka, it was estimated to be around 12% in 2012 (personal communication, UTH Dept. of Obstetrics and Gynaecology). Women of African descent are said to be more likely to develop preeclampsia than those of the Caucasian population [11]. This has been attributed to genetic factors [11].

1.1 Pulse Wave form and Velocity

A typical pulse waveform contains an augmentation pressure (AP) which represents the pressure from systolic shoulder to the peak. Its index, (augmentation index (Aix)), is calculated as the difference between P_2 and P_1 expressed as percentage of the pulse pressure.

These indices are all markers in cardiovascular measurements. However, these have not been documented in pregnant women of African descent. This study seeks to explore this aspect.

1.2 Differences in Arterial Pulse Wave Propagation

The pulse waveform's shape is said to be a result of the summation of a direct wave and a reflected wave, both of which propagate along the arterial tree. The shape and magnitude of these waves are able to change as the characteristics in the structure of the arteries change. The arrival of the reflected wave is dependent on the compliance of the arterial wall. In a compliant elastic artery the reflective wave is said to travel rather slowly and returns in late systole [1]. However, in a stiff artery, typical of hypertensive disorders, the reflected wave is said to travel faster resulting in a greater amplification of the peak (augmentation pressure) and returns in early systole consequently increasing the aortic pressure [1]. This has implications on approaches to treatment modalities. While this has been noted in both primary and secondary hypertension, very little literature exists on the waveform and magnitude of PWV in PIH.

2. MATERIALS AND METHODS

This cross sectional study included all pregnant women between the age of 18-45 years presenting to the UTH department of obstetrics and gynecology for a routine antenatal clinic visit during the study period (April to June 2014) who met the eligibility criteria and gave consent to participate. Excluded were pregnant women younger than 18 years and older than 45 years or women with chronic hypertension, diabetes mellitus and any known cardiovascular pathology.

2.1 Socio-Demographic Data

All consenting participants were interviewed to obtain socio-demographic data and health information such as maternal age, marital status, gestational age, smoking status or exposure to tobacco smoke, history of diabetes mellitus or use of either hypoglycaemic agents, history of hypertension or use of anti-hypertensive medication, alcohol consumption, physical exercise, family history, history of other cardiovascular conditions and/or use of other medications.

2.2 Anthropometric Measurements

Body height was measured to the nearest 0.1 cm using the Seca Brand 214 Portable Stadiometer (Seca gmbh & Co. kg Humburg, German). Participants were asked to remove their foot and head gear and with their heels against the back board looking ahead, measurements were taken. Weight was measured to the nearest 0.1 kg using the Heine Portable Professional Adult Scale 737 (Seca gmbh & Co. kg Humburg, German). Participants were again asked to stand still with their face forward, and arms on the sides of the body. The length from the carotid artery (neck) to the radial artery recording sites was measured using a Figure-Finder tape measure.

2.3 Pulse Wave Velocity Measurements

After a 15 minute rest, peripheral systolic and diastolic BP was measured three times on the right arm whilst seated using an Omron M6 Comfort automatic BP monitor. The last two BP measurements were then averaged. Measurements were taken at 3 minute intervals. Participants were then asked to lie in the left lateral position to avoid vena cava compression by the uterus for another period of rest of 10 minutes. The Complior analyse was utilised to measure PWV. The PWV measurement involved applying non-invasive piezoelectric sensors over the skin after palpating for the carotid artery on the neck and the radial artery on the wrist. Other information measured with this software included: The augmentation index (Aix), a composite measure of systemic arterial stiffness, central systolic blood pressure (cSBP), central diastolic blood pressure (cDBP), central pulse pressure (cPP) and mean central arterial blood pressure (cMAP). During measurement, the women were requested not to move or speak. All measurements were taken by the same observers for all participants.

3. RESULTS

3.1 Anthropometric Measurements

A total of 26 women participated in this study. Of these, 14 were normotensive, 12 were newly diagnosed hypertensive participants.

Both groups recorded similar age ranges with means of 27 and 31 years for the normotensive and hypertensive participants respectively. The mean gestational age was also similar in both blood pressure groups. The body mass index (BMI) for all women showed that they were overweight. Details of these characteristics are outlined in Table 1.

3.2 Blood Pressure Measurements

Table 2 shows an aggregation of both peripheral (brachial, b) and central (c) blood pressure results. Peripheral BPs recorded an average of 93/59 mmHg amongst the normotensives and 124/78 mmHg amongst hypertensive participants ($p < 0.001$). The average central BP readings from Carotid-Radial recordings of the Complior were 86/59 mmHg amongst the normotensive participants while the hypertensive participants recorded average central BPs of 119/78 mmHg which were also significantly higher ($P < 0.001$).

3.3 PWV Measurements

Table 3 shows carotid-radial recordings made for PWV, augmentation pressures (AP) and augmentation index (Aix). crPWV recorded 9±4 m/sec for normotensives and 13±7 m/sec for hypertensive participants ($P < 0.05$).

3.4 Augmentation Pressure

The augmentation pressure averaged 4±5 mmHg for normotensives and 9±8 mmHg for the hypertensive participants ($p<0.05$).

3.5 Augmentation Index

For the augmentation index (Aix), normotensive participants recorded 1±22% and the hypertensive participants recorded 16±23% ($p<0.05$).

3.6 Pulse Waveform

The Tsys period was completed before the T1 period elapsed. Thus consequently P2 preceded P1.

T1 period was completed before the Tsys period elapsed. Consequently P1 preceded P2, indicating that the return of the reflected wave occurred in early systole causing an elevation in the systolic pressure and consequently augmentation pressure.

Normotensive participant showed that Tsys (the timing of the systolic wave) period was completed before the T1 period elapsed (timing of the reflected wave). This indicates that the reflected wave arrived in late systole (type C wave). However for hypertensive participants T1 period was completed before the Tsys period elapsed showing that the reflected wave came early in systole (type A). Thus, AP was larger in the hypertensive participant as a result of an increased systolic pressure.

The relationship between central and peripheral diastolic pressures and crPWV was investigated using Spearman's correlation. There was a strong positive correlation between central and peripheral diastolic pressures and crPWV rho= 0.626 and 0.590 respectively at a significance level of 0.01 (n=26).

A moderate positive correlation was seen between central and peripheral systolic pressures and crPWV rho=0.396 and 0.423 respectively at significance of 0.05 level with shared variance of 15.68% and 17.89% respectively.

4. DISCUSSION

4.1 Demographical Differences

The age groups of all consenting participants showed no significant difference and thus were comparable ($P=.14$). This is notable as age is a determinant of arterial stiffness and consequently BP both of which increase with age [13]. This is attributed amongst other things to the decrease in nitric oxide synthesis and elastin fragmentation and degradation leading to the loading of collagen fibers in the arterial tree.

Table 1. Baseline characteristics of study groups

	Normotensive (NTN) n=14	**Hypertensive (HTN) n=12**	***P value***
Maternal age (yrs)	27±6	31±5	**NS*
Gestational age (weeks)	26±12	29±7	**NS*
Body Mass Index (kg/m^2)	27±5	29±4	**NS*
Weight (kgs)	72±15	77±15	**NS*
Height (cms)	164±8	154±32	**NS*

*Values are given as means ± standard deviation. Asymptotic significances displayed across all study groups. The significance level is 0.05. *NS = Non-significant*

Table 2. Peripheral (brachial, b) and central (c) blood pressure

Brachial BP	**NTN**	**HTN**	***P value***
bSBP (mmHg)	93±9	124±18	*< 0.0001*
bDBP (mmHg)	59±8	78±10	*<0.0001*
bPP (mmHg)	34±6	46±12	*<0.0001*
bMAP (mmHg)	70±8	93±12	*<0.00*01
Central Carotid–Radial (C-R) BP measurements			
cSBP (mmHg)	86±10	119±18	*<0.0001*
cDBP (mmHg)	59±8	78±10	*<0.0001*
cMAP (mmHg)	68±7	90±13	*<0.00*01
cPP (mmHg)	27±11	37±15	*<0.05*

bSBP=brachial systolic BP, bDBP= brachial diastolic BP, bPP=brachial pulse pressure, bMAP= brachial mean arterial pressure, cSBP=central systolic BP, cDBP= central diastolic BP, cMAP= central mean arterial pressure, cPP= central pulse pressure. Values are given as means ± standard deviation. The significance level is 0.05. Mann-whitney u test was used

Table 3. Pulse Wave velocity (PWV), augmentation pressure (AP) and augmentation index (Aix)

	NTN	**HTN**	***P value***
crPWV (m/sec)	9±4	13±7	*< 0.05*
AP (mmHg)	4±5	9±8	*<0.05*
Aix (%)	1± 22	16±23	*<0.05*

*AP= augmentation pressure, Aix= augmentation index, PWV= pulse wave velocity. Values are given as means ± standard deviation. The significance level is 0.05. Mann-whitney u test was used. * shows significant difference*

All the participants were of comparable height, weight, and body mass index. The height of an individual defines the distance travelled by blood to particular reflection points. 'The shorter the distance travelled, the greater the amplitude of the reflected pulse wave' [14]. Increases in fatty deposits (plaques) on the arteries leads to arterial stiffening and narrows blood vessels compromising its flow. Thus weight and height can be confounders to arterial stiffness. However, because there were no significant differences amongst these categories, participant groups were considered comparable in this study.

4.2 Blood Pressure

This study joins many other studies in establishing significant differences in peripheral and central blood pressures of normotensives when compared to hypertensive participants. These increases in blood pressure are said to be due to the structural changes that occur on the vascular lining of these vessels. This suggests that a relationship exists between blood pressure and PWV regardless of whether the arterial stiffness is a cause or consequence of raised blood pressure.

4.3 Augmentation Index (Aix)

Aix, a surrogate measure of the reflected pulse wave, is an important cardiovascular measure because it is able to distinguish between the effects of different vasoactive medications which may not be appreciated using PWV [15]. A significant difference was found in Aix between hypertensive and normotensive participants. This suggests that significant arterial stiffness was prevalent and consequently the reflected wave returned before ventricular ejection in the hypertensive participants.

4.4 Pulse Wave Velocity (PWV)

This study has attempted to establish mean PWV values in normotensive and hypertensive pregnant women of African descent. crPWV was significantly different between these two groups, being higher in the hypertensive group. This suggests that the proposed endothelial dysfunction had occurred along the carotid-radial segment which spans the subclavian, brachial, and radial arteries [16], resulting in significant arterial stiffness. This increased stiffening resulting in a decrease in diameter of the vessel, causes increases in blood flow and consequently blood pressure. Central blood pressures, often described as a better predictor of cardiovascular events, and peripheral blood pressures correlated positively with PWV (cSBP; rho=0.396, cDBP; rho=0.626, bSBP; rho=0.423, bDBP; rho=0.590), conclusively demonstrating that hypertensive participants had higher PWV than normotensive participants.

With the reduction in systemic vascular resistance (SVR) that occurs in the normal adaptation of pregnancy, a reduction in both systolic and diastolic blood pressures is expected with diastolic pressure being more reduced [17] in both groups. This was evidenced by the strong positive correlation seen between crPWV and diastolic blood pressure (rho=0.626).

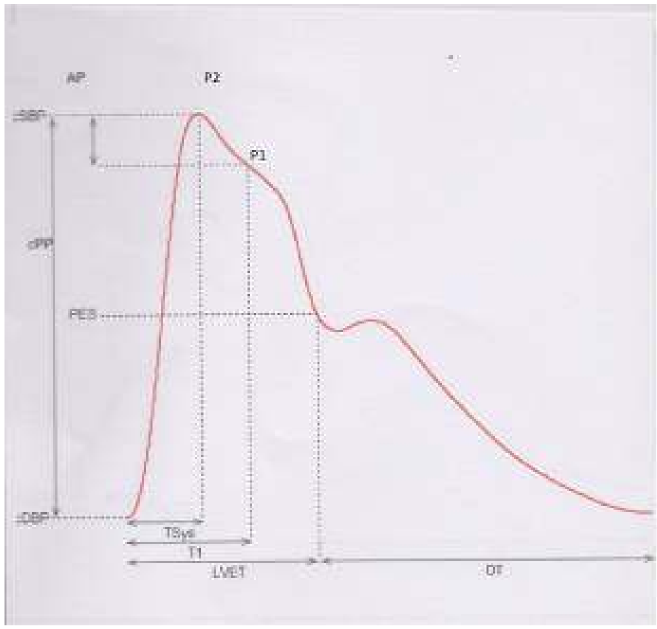

Fig. 1. A recording of pulse waveform for a normotensive participant

cSBP= central systolic BP,cPP=central pulse pressure, cDBP=central diastolic BP, AP= augmentation pressure, PES= end systolic BP, Tsys=timing of the systolic wave, T1= timing of the reflected wave, LVET= left ventricular ejection time, DT= diastolic time, P1= first systolic peak, P2=second systolic peak

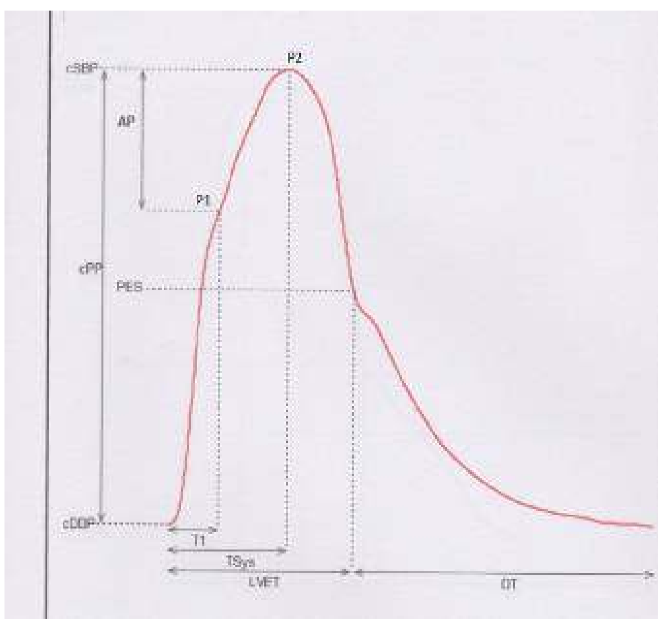

Fig. 2. A recording of pulse waveform for a hypertensive participant

cSBP= central systolic BP,cPP=central pulse pressure, cDBP=central diastolic BP, AP= augmentation pressure, PES= end systolic BP, Tsys=timing of the systolic wave, T1= timing of the reflected wave, LVET= left ventricular ejection time, DT= diastolic time, P1= first systolic peak, P2=second systolic peak

The waveforms generated were typical to those described in literature [11,6,18]. A C-type wave was generated in normotensive participants. This wave depicts the return of the reflected wave in late systole after ventricular ejection has ceased, characteristic in young adults (>30 years) [1,19]. In hypertensive pregnant individuals, either an A or B type wave is said to be generated. In these waveforms, the reflected wave arrives early in systole during ventricular ejection, and the timing of the reflected wave (T1) is shorter than the systolic wave (T2) [1,19]. Similar types of wave forms were recorded in this study for both groups. Amplification of the systolic pressure is also seen in these participants. This can be seen by an increase in the AP. Results show a hypertensive participant with a type A wave because their Aix is above 12% (Aix =31.04%).

In more severe cases of PIH, increased arterial stiffness, increased endothelial dysfunction resulting in proteinuria and increased blood pressures are expected. However, amongst the participants included in this study, 11 of the 12 hypertensive participants had 0-trace amounts of protein in their urine. Hence comparisons could not be made between arterial stiffness and proteinuria.

4.5 Limitations

Characteristics of PWV were not measured longitudinally throughout pregnancy and after delivery, to assess the genesis and persistence of the arterial stiffness. Variations are seen in aortic stiffness throughout the gestational period with its lowest in the second trimester and its' highest in the third during normal pregnancy [6]. This change in arterial stiffness is said to be due to the remodelling of blood vessels that occurs in pregnancy resulting in increases in cardiac output, plasma volume and decrease in vascular resistance. This remodelling ceases once birth occurs.

5. CONCLUSION

Arterial stiffness, as evidenced by carotid-radial pulse wave velocity, augmentation pressure and augmentation index, increases significantly in PIH. This has been attributed to the endothelial dysfunction that occurs in PIH. There are distinct differences in the pattern of the carotid-radial pulse waveform in PIH. In normotensive individuals the reflected wave returns to the ascending aorta after ventricular ejection. In contrast, hypertensive individuals had their reflected wave returning during ventricular ejection causing an augmentation of the systolic pressure and consequently an increase in the augmentation pressure. With the distinct vascular changes that occur in PIH more studies need to be conducted as PWV may be used as an early predictor of women at risk of developing PIH and as a factor for initiating and monitoring treatment.

ETHICAL APPROVAL

The study protocol was approved by ERES Converge IRB.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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