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Duplex Carotid Scan in Hypertensive Patients at the Rivers State University Teaching Hospital: PORT Harcourt. Nigeria

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Abstract
Background: *Carotid vascular scan has proven a very valuable tool in assessing atherosclerotic source of large vessel thromboembolic stroke. The stroke burden is high in Nigeria and globally, it corresponds to the prevalence of hypertension. Hypertension is the leading cause of Ischemic stroke. The relationship of hypertension and carotid vascular parameters will be an interesting study. Duplex carotid vascular scan is non-invasive, cheap, and readily available. This study set out to evaluate the effect of hypertension on carotid vascular parameters by comparing carotid artery parameters between a hypertensive population and that from normal individuals.*
Method: *Hypertensive's, nondiabetics attending clinics at the Rivers State University Teaching Hospital, from January 2021 to October 2021, were recruited for the study and carotid artery parameters from normal, non-hypertensive and non-diabetic data was compared after carefully matching for body mass indices. A protocol was developed with duplex vascular parameters of the left common carotid (LCC), right common carotid artery (RCC), left the internal carotid (LIC), and right internal carotid artery (RIC). This protocol was used to assess the hypertensives and values obtained were compared to that of a normal Nigerian Population. Peak Systolic Velocity (PSV) End diastolic velocity (EDV) the Resistivity index (RI) the ratio of PSV /EDV (S/D), the vessel diameters and carotid Intima Media Thickness (cIMT). Data was collected on excel spreadsheet and analyzed using Statistical Package for the Social Sciences (SPSS) 23.*
Results: *Eighty-one (81) hypertensive who met the inclusion criteria and data for fifty (50) apparently normal individuals was utilized. The mean age ±SD for the hypertensive population: normal was 57.26 ± 10.53yrs: 48.67 ± 7.11yrs. Mean systolic blood pressures ±SD of hypertensive population: normal was 167.46 ± 17.73mmHg: 129 ± 11.29mmHg, mean diastolic blood pressure ±SD for hypertensive: normal was 88.63 ± 12.57mmHg: 76.04 ± 8.15 mmHg. There was no significant difference in their BMI.*
The left common carotid showed statistically significant difference in its IMT, and diameter when compared with the normal population and the left internal carotid showed significantly larger vessel diameter and increased IMT when compared to the normal parameters. On the right there was a significantly larger vessel diameter of the RCC and increased IMT for both RCC and RIC. Carotid doppler velocities showed higher EDV of the left internal carotid artery and S/D ratio was significantly higher in the LCC, LIC and RCC arteries.
Bivariate analysis using the Pearson correlation coefficient revealed a negative correlation at significant at 0.01level (2 tailed) between SBP and the PSV (-441, 0.002) and EDV (-365, 0.011) of the Left Internal Carotid artery. There was also a positive correlation of the EDV and the left internal carotid IMT (0.385,0.009). However on the RCC and RIC there was no correlation with blood pressures in our study population. The blood pressure affected the left internal Carotid artery more than the left common carotid artery.
Forty-three (53.09%) of the hypertensive population had left common cIMT > 0.09cm. Forty-one (50.0%) had left internal cIMT >0.09cm. Forty-five (55.56%) of the hypertensive group on the right common cIMT > 0.09cm and right carotid 38(46.91%) had cIMT > 0.09cm. Twelve (12) and (15.39%) had plaques of varying

echogenicity. A notable finding was a higher peak velocity relative to end diastolic velocity (high SD ratio) noted at points of stenosis which could be doppler pointer to stenosis in the especially in the echolucent plaques.
Conclusion: *Hypertension affects the structure and blood flow across the carotid vessels significantly as shown in its impact on PSV, EDV and S/D and there is need to evaluate the role of doppler parameters as surrogates of cardiovascular markers.*
Keywords: *Carotid, Hypertensive, cIMT, cS/D ratio, Nigerian, Stroke, cPSV, cEDV*

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I. Introduction:

Hypertension is a leading cause of stroke globally; it is the highest preventable risk factor for stroke.[1] Hypertension has been implicated as a risk factor for all three types of strokes ischemic, intracerebral, and subarachnoid hemorrhage. In both developed and developing countries ischemic stroke is the leading type of stroke [2-6] Duplex vascular ultrasound has improved the use of non-invasive testing in evaluating vascular diseases [7]. Findings at vascular ultrasonography have been corroborated with findings at arteriography [8]. Vascular ultrasound is very useful in plaque definition and morphology. [9].

Atherosclerosis in large vessels have been implicated as the source of ischemic stroke [10]. 15% of strokes have been attributed to large vessel atherosclerosis [11-13] Another study attributed 8% of strokes to atherosclerotic disease from extracranial carotid arteries [14]. An important point of note with large vessel atherosclerotic disease is the high recurrence rate of stroke with large vessel disease [10]. Internal carotid prompt end atherectomy has been shown to prevent recurrence of stroke [15] Extracranial internal carotid artery stenosis has been implicated as the most common cause of large vessel stroke [14] and its management has been an important area of interest by large trials [16,17]

Carotid doppler ultrasonography has been used to evaluate hypertensives in previous studies. A study proposed that vessel integrity; diameter and velocity except is maintained except in the presence of a plaque [18]. This study set out to understand the crucial effect the hypertension has on extracranial large vessel atherosclerotic diseases by comparing findings from a hypertensive population with that of a normal population.

II. Method.

It was a prospective, observational, cross-sectional study aimed at assessing the impact of hypertension on the carotid vessels, by comparing doppler carotid vascular parameters found in a hypertensives population with values obtained for a healthy population(controls). It was a hospital-based study, carried out in Rivers State University Teaching Hospital from January 2021 to October 2021. Ethical clearance was obtained from the Ethics committee of the hospital and consent obtained from patients after fully counselling them on the procedure.

Sample Size: The stroke prevalence in a Nigerian community-based study was given as 1.31/10000 [19] However, a study from community in Rivers State obtained a crude prevalence of 8.51/1000 for stroke [20]. Computing the sample size with the formula

$$n= Z^2 (pq)/e^2$$

Where n = minimum sample size

Z = 1.96 at a CI of 95%, q= 100 – p, p= Prevalence rate 0.851%, at a margin sampling error at 5%. (Substituting in the formula above)

$$n = \frac{(1.96)^2(100-0.851* 0.851)}{(0.5)^2}$$

$$\frac{9.29}{0.25}$$

n= 37.

Eighty-one was used as the sample size as hospital prevalence is expected to be higher.

Hypertensive population: The study recruited 81 hypertensives who met the inclusion criteria and consented to be part of the study; hypertensives aged 40yrs and above, who had their blood glucose assessed to exclude diabetes. Body mass index (BMI) was computed after measuring their weights and heights, pulse and blood pressure was measured using a digital sphygmomanometer before the ultrasound procedure.

Controls: Data from previous studies was used as the Controls. Carotid artery parameters for CCA and ICA was retrieved for fifty normal (50) subjects, aged 40yrs and above after carefully matching for BMI.

Ultrasound procedure: Study was carried using the Sonoscape SS1 8000, both with an ultrasonic duplex scanner with a 752 (5MHz – 15 MHz) frequency linear array probes which provided simultaneous real time 2 D arterial images and single gated pulse wave velocity for doppler interrogation. A proforma was developed with patient's biodata, weight, and height: from which BMI was computed, pulse rate, symptoms and clinical

findings and made to include a protocol for CCA and ICA which measured vessel diameter, intima media thickness, peak systolic velocity (PSV), end diastolic velocity (EDV) the resistivity index (RI) and the ratio of the PSV/EDV (S/D ratio). The patients were made to lie supine and asked to extend their necks and turn their face to the opposite side of the carotid vessels to be examined. Carotid examination was performed by placing the scan head on the neck along the axis of the carotid vessels. Examination was started proximally in transverse and follow distally to the bifurcation this was to assess the course and the presence of any intimal thickening or plaque. This was repeated in longitudinal plane. Color flow superimposed on 2D gray scale to assess patency highlight possible stenosis and importantly to assess the doppler velocity. In measuring velocity care was taken to obtain sample volume from the center of the vessel and not towards the wall to prevent turbulence along vessel wall interfering with reading. The readings were taken about the mid common carotid and internal carotid artery. Two successive readings were taken, and the mean computed for each carotid artery variable.

III. Results:

Eighty-one (81) :47 (51.28%) males and 34(48.72%) females made up the hypertensive population and data was retrieved from 50 (fifty): 27(54%) males and 23(43%) females after carefully matching for BMI. The mean parameters for Hypertensive: Normal, (Age: 57.26 ±10.53yrs:48.67 ±7.11yrs), (SBP:167.46 ± 17.73mmHg:129.33 ±11.29mmHg) (DBP: 88.63±12.57mmHg: 76.04 ± 8.15mmHg) were significantly higher in the hypertensive population and normal (see Table 1). For the hypertensive population the males and females compared favorably with no significant differences in age, BMI, Pulse rate, systolic and diastolic blood pressure. (See Table 2).

Majority of the patients were asymptomatic however there were histories of TIA, syncope, and previous strokes in some of the patients evaluated (See table 3).

There were significantly higher values, on paired T test analysis of the LCC mean; diameter, IMT and SD (P value 0.05) in the hypertensive population when compared to the mean. The mean of the LIC diam (.00), IMT (.00), EDV and SD (P value 0.05) was also significantly higher in the hypertensive group than the mean for the normal population.

On the right, the RCC mean values for IMT, Diam was significantly higher in the hypertensive population than that of the normal population. and RIC IMT was significantly higher in hypertensive than the normal (see Table 4).

Bivariate analysis using the Pearson correlation coefficient revealed a negative correlation at significant at 0.01 level (2 tailed) between SBP and the PSV (-441, 0.002) and EDV (-365, 0.011) of the Left Internal Carotid artery. There was also a positive correlation of the EDV and the left internal carotid IMT (0.385,0.009). However, on the RCC and RIC there was no correlation with blood pressures in our study population. The blood pressure affected the left internal Carotid artery more than the left common carotid artery.

Forty-two (52.4%) of the hypertensive population had LCC IMT> 0.09cm [21]. Forty-one (50.0%) had LIC IMT >0.09cm. 54% of the hypertensive group on the RCC showed IMT > 0.09cm. 12 (15.39%) had plaques. Six (6) of the plaques was located in the LIC artery causing a 20 -80% stenosis. In two subjects the plaques were found in both the LCC and the LIC (see figure 1). One (1) showed a RCC plaque. Isolated LCC plaque was discovered in two (2) and three (3) had isolated RIC plaques. Most of the plaques were echogenic plaques. In the study population most were symptomatic 2 had TIA, 2 had a previous stroke. Carotid bruit was discovered in one patient with RCC plaque whilst others had headaches and dizziness.

A notable finding was a higher peak velocity relative to end diastolic velocity (high SD ratio) noted at points of stenosis which could be doppler pointer to stenosis in the echolucent plaques, The loss of spectral window is another doppler sonographic guide to stenosis.

IV. Discussion

Carotid doppler has proven a very valuable tool, in assessing large vessel source of atherosclerotic disease and thrombo-embolism in stroke patients. Sources of thromboembolism varies in the etiopathogenesis of Ischemic stroke. Studies have shown cardiac sources [22] and other large vessel sources [23]. Internal carotid artery atherosclerotic disease has however become an important area for trials with the aim of optimizing care in these group of patients. There are studies locally [24] (in Nigeria) that has evaluated carotid doppler findings in hypertensive, there is no doubt that these studies have given attention to an important surrogate marker for cardiovascular diseases: cIMT. However, this study has shed some light on carotid doppler findings in addition to cIMT changes and diameter changes with duplex carotid vascular scans.

Carotid Vessel Diameter: Hypertensives have been shown to have significantly larger vessels when compared to the normal population. [25] We studied the diameter of four carotid artery vessels, the LCC, RCC, LIC and the right internal carotid by doppler gray scale, longitudinal cut. Measuring the distance between the intima of both vessel walls, showed significantly larger vessel diameters in all four vessels studied.

The mechanism by which hypertension causes dilatation of vessel recorded in other large vessels like the femoral arteries[26] a prospective study[27] that tried to offer an explanation, for the dilation of the carotid artery by hypertension; compared the diameter- pressure curves of carotid arteries and femoral vessels of hypertensive and normotensive subjects and discovered that the carotid artery dilation was dependent on distending pressure, in that same study a different mechanism was proposed for the femoral artery dilatation; the femoral dilatation was attributed to geometric change in vessel caliber. This would mean that controlled blood pressure would imply an absence of dilatation. While this may offer an explanation for the significant increase in diameter in our study population; which studied a hypertensive group with poor blood pressure control: Mean ± SD of SBP:167.46 ± 17.73mmHg:129.33 ±11.29mmHg, and saw a systolic blood pressures as high as 200mmHg and diastolic as high as 120mmHg. However, based on the theory put forward by the study of Armentano et al, this study was expected to have shown a correlation with either systolic or diastolic blood pressures, with vessel diameter but there was no correlation by bivariate analysis. Therefore, a mechanism whereby the initial effect could be the pressure effect, which could over time result in geometric changes or a combination of both factors is a possible mechanism of carotid artery vasodilatation in hypertensives, corroborated in this study

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***c*IMT:** The impact of high blood pressure in hypertensive study on IMT was a very notable finding and has been seen in other studies [28-30]. The effect of blood pressure on IMT has not been shown to be affected by the duration of hypertension[31]. Carotid IMT is important surrogate of cardiovascular disease and corresponds to atherosclerotic disease in other large vessels. It has shown a linear relationship with hypertension [32].

LCC IMT in the index study showed a positive correlation with hypertension. There was also significant difference between IMT across most of the carotid vessels between the hypertensive group and data from controls. In some of our hypertensive subjects, marked IM hypertrophy was noted (see table 6 and figure 3). Clinical trials have shown the regression or slow progression of IMT in clinical trials with therapy [32] Care must be taken to differentiates IMT from plaques and in some cases intimal media hyper trophy or hyperplasia can co-exist. IMT is a double-line pattern visualized by echography on both walls of the CCA in a longitudinal image. Two parallel lines, which consist of the leading edges of two anatomical boundaries, form it: the lumen-intima and media-adventitia interfaces [33]. While Plaques are focal structures encroaching into the arterial lumen of at least 0.5 mm or 50% of the surrounding IMT value or demonstrates a thickness >1.5 mm as measured from the intima-lumen interface to the media-adventitia interface [34].

Doppler Flow Velocities and Ratios: This study showed the association between systolic blood pressure and Diastolic blood pressure and carotids artery PSV and EDV. The PSV and EDV showed an inverse relationship with the systolic and diastolic blood pressures in the left carotid artery. This is like a study that evaluated the impact of hypertension on cognitive function:[35] the study deployed brain blood flow by evaluating the relationship between SBP and DBP in hypertensives and noted an inverse relationship. This study also noted the significance of the ratio of the PSV to the EDV: the S/D ratio. The S/D ratio was relatively widened in stenotic vessels with higher PSV relative to EDV (see figure 1). The mechanism of this is not clear. Future studies to understand this is encouraged. The spectral window is obscured at doppler evaluation of stenotic sites.

Conclusion: Hypertension affects the structure and blood flow across the carotid vessels significantly as shown in its impact on PSV, EDV and S/D and there is need to evaluate the role of doppler parameters as surrogates of cardiovascular markers

FIGURE 1: IDENTIFICATION OF ECOLUCENT PLAQUE USING SD RATIO

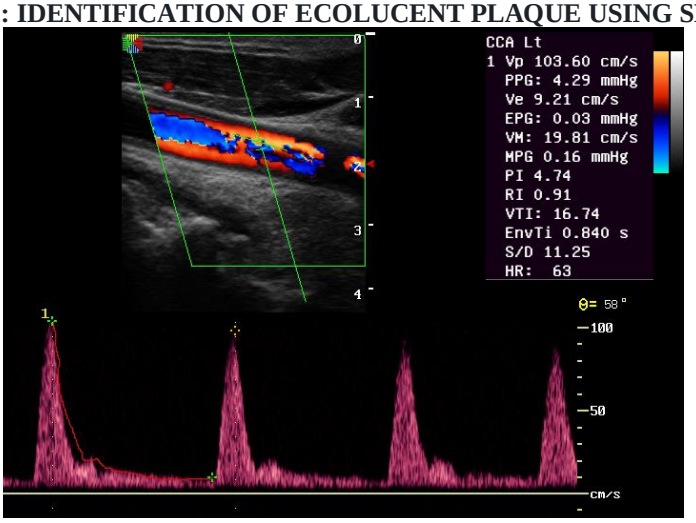


FIGURE 2: HETEROGENOUS PLAQUE

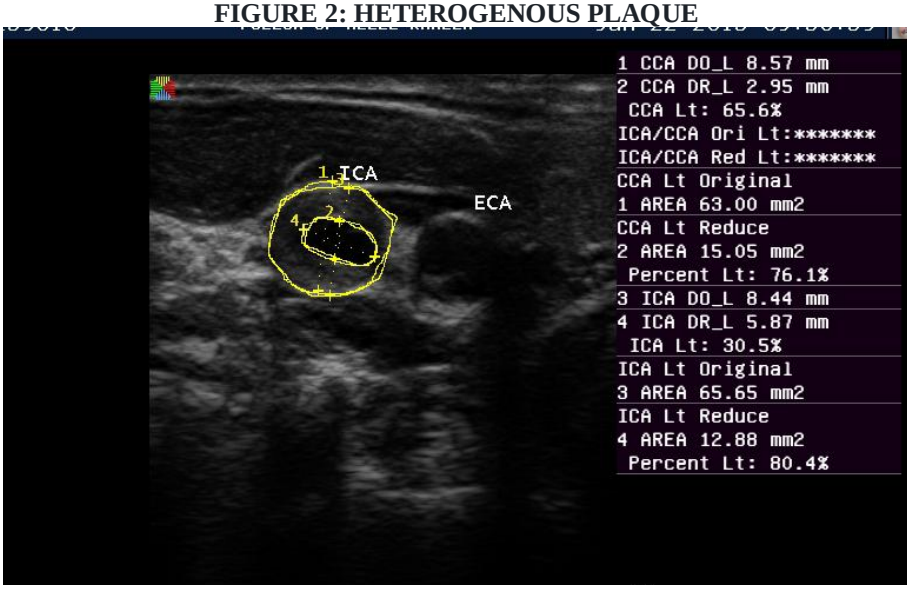


FIGURE 3: MARKED HYPERTROHY AFFECTING BOTH ICA AND ECA WITH PLAQUE DEVELOPMENT IN THE ECA

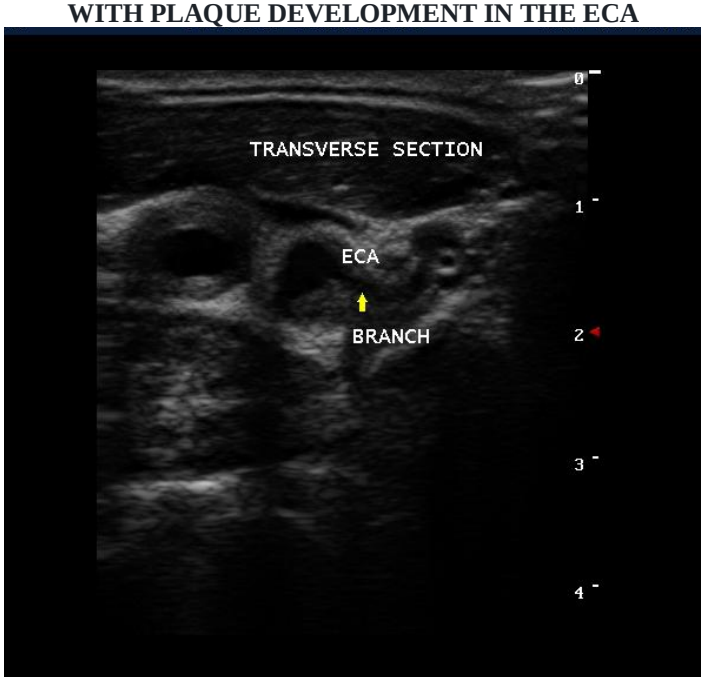


TABLE 1 : COMPARISON of CLINICAL PARAMETERS BETWEEN HYPERTENSIVES AND NORMAL

Clinical Parameters n	*Normal 50*	*Hypertensives 78*	*Std. Error Mean*	*T*	*Sig. (2-tailed)*
AGE	48.67 ±7.11	57.26 ±10.53	1.78	4.82	.000
BMI	25.17 ± 3.57	26.44± 6.34	1.09	1.16	.252
PULSE	77.90±13.12	80.73 ± 9.89	2.19	1.29	.202
SBP	129.33 ±11.29	167.46 ± 17.73	3.28	11.63	.000
DBP	76.04 ± 8.15	88.63±12.57	2.34	5.37	.000

Significant at P value ≤ 0.05

TABLE 2 : COMPARISON of CLINICAL PARAMETERS BETWEEN MALE AND FEMALE HYPERTENSIVES

Clinical Parameters	*Male Hypertensives*	*Female Hypertensives*	*Std. Error Mean*	*t*	*Sig. (2-tailed)*
AGE	58.74 ± 11.13	56.05 ±10.49	2.24	-1.20	0.24
BMI	26.46 ± 6.12	25.62 ± 7.17	1.48	-.57	0.58
PULSE	81.97 ±11.80	80.68 ± 7.84	2.43	-.53	0.60
SBP	173.316 ±17.51	164.47 ± 15.16	3.97	-2.23	0.03
DBP	90.13 ±12.03	87.11 ±11.39	2.58	-1.18	0.25

Significant at P value ≤ 0.05

TABLE 3: CLINICAL FINDINGS

Clinical Findings	**Frequency**	**Valid Percent(%)**
Asymptomatic	**48**	**59.3**
Amnesia, and Headache	1	1.2
Bradycardia	1	1.2
Carotid Bruit	1	1.2
Chest Pain /Dizziness	1	1.2
Dizziness	2	2.5
Full Work-Up	1	1.2
Headache	3	1.2
Headache/Dizziness	5	1.2
Insomnia	3	1.2
Pedal Swelling	1	1.2
Previous CVD	3	3.7
Syncope	10	8.6
TIA	4	4.9
Total	81	100.0

TABLE 4 : COMPARISON of LEFT CAROTID ARTERY PARAMETERS BETWEEN HYPERTENSIVES AND NORMAL

Left Carotid artery Parameters	*Normal*	*Hypertensives*	*Std. Error Mean*	*t*	*Sig. (2-tailed)*
LCC/Diameter	0.69 ±.07	0.74 ±0.11	.02	3.00	.00
LCCIntima Media Thickness	0.07±0.02	0.13±0.04	.01	4.70	.00
LCC Peak Systolic Velocity	60.40±24.87	67.37 ± 22.25	4.57	1.53	.13
LCC End Diastolic Velocity	31.21 ± 22.25	25.93±18.35	4.39	-1.20	.23
LCC Resistivity Index	0.51±0.19	0.61 ± 0.19	.042	1.90	.06
LCC SD	2.54 ±1.15	3.12 ±1.54	.29	1.98	.05
LIC Diameter	0.73±0.14	0.82±0.19	.03	-2.90	.01
LIC Intima Media Thickness	0.06±0.02	0.13±0.01	.02	3.38	.00
LIC Peak Systolic Velocity	53.52±266	54.00 ± 20.11	4.81	.10	.92
LIC End Diastolic Velocity	28.60±26.05	18.69 ±16.20	3.71	-2.67	.01

LIC Resistivity Index	0.54±0.23	0.60±1.20	.05	1.26	.21
LIC SD	2.94±2.01	5.35±13.69	2.08	-1.16	.00
RCC/Diameter	0.68±0.12	0.75± 0.13	0.02	3.30	.00
RCCIntima Media Thickness	0.06 ±.017	0.12±0.11	0.02	3.43	.00
RCC Peak Systolic Velocity	50.93± 20.85	107.33 ± 30.58	44.17	1.28	0.21
LCC End Diastolic Velocity	25.00 ± 20.04	20.73 ±15.07	3.25	1.31	0.20
RCC Resistivity Index	0.62±0.17	0.99 ± 0.28	0.40	-0.91	0.37
RCC SD	2.96±1.23	3.27 ±1.90	0.34	0.93	0.36
RIC Diameter	0.75± 0.17	0.77± 0.17	0.04	0.58	0.57
RIC Intima Media Thickness	0.06 ±0.01	0.10 ±0.05	0.01	5.06	0.00
RIC Peak Systolic Velocity	52.60±24.90	54.12±28.55	4.92	0.31	0.76
RIC End Diastolic Velocity	26.46±22.44	24.93 ± 13.94	5.17	-0.30	0.77
RIC Resistivity Index	0.56±0.20	0.57±0.25	0,05	0.16	0.87
RIC SD	2.81±0.78	3.22±2.29	0.48	0.85	0.40

Significant at P value ≤ 0.05

TABLE 5: CORELATION BETWEEN LIC PARAMETERS AND SBP & DBP

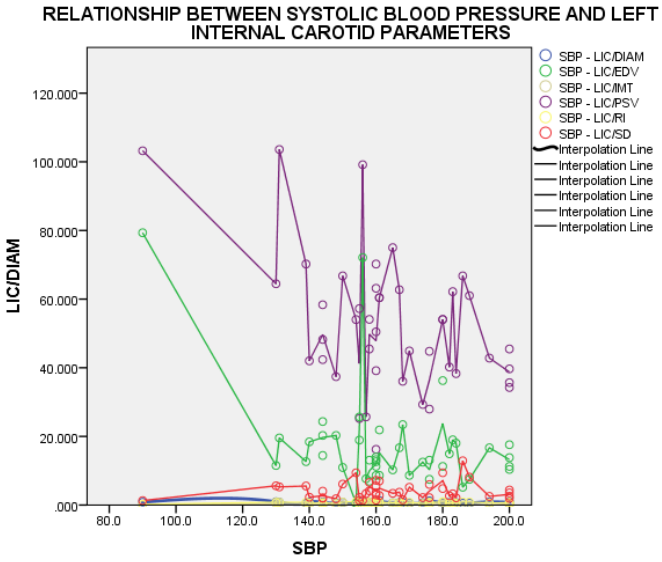
	Blood Pressure	**LIC IMTLIC**	**PSV LIC EDV**	
Pearson Corelation Coefficient	SBP	.181	-.441**	-.365*
Significance Level		.234	.002	.011
Pearson Corelation Coefficient	DBP	.385**	-.280	-.111
Significance Level		.009	.054	.454

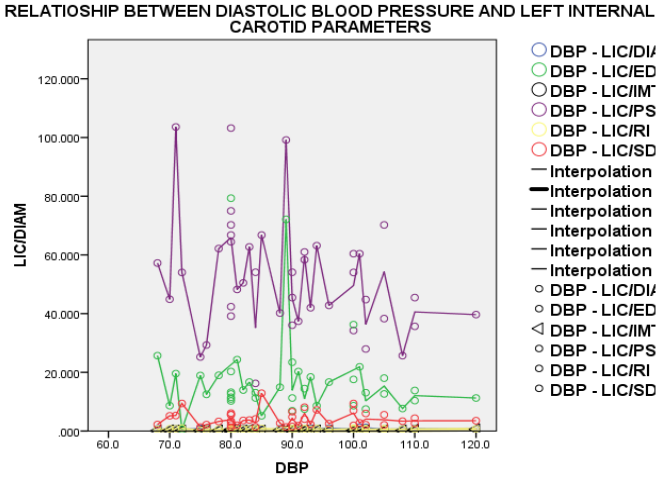
**Correlation is significant at the 0.01 level(2 tailed)
*Correlation is significant at the 0.05 level(2 tailed

TABLE 6: *c*IMT MEASUREMENT ACROSS HYPERTENSIVES

CAROTID VESSELS	IMT ≤ 0.09	**IMT 0.09 – 1.9**	IMT ≥2
Right Common Carotid	57	19	4
Right Internal Carotid	59	20	3
Left Common Carotid	49	30	2
Left Internal Carotid	58	17	6

***c*IMT= Carotid Intima Media Thickness.**





Primary Health Care Management **Board** (RSPHCMB) for permission and ethical clearance to carry out this study.

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Compliance and Ethics Guidelines: The study was in keeping with best research practices: ethical clearance was sought from the ethics committee of the **Rivers State University Teaching Hospital**s and consent obtained from each subject after proper education on the procedure.

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