J. Cardiovasc Disease Res., 2020; 11(4): 249 – 254
A Multifaceted Peer Reviewed Journal in the field of Cardiology
www.jcdronline.org | www.journalonweb.com/jcdr
P – ISSN: 0975 – 3583 Online: 0976 – 2833

# Relationship Analysis between Flow Mediated Dilatation of Brachial Artery and Hypertension along with Other Related Parameters

**[1*]V.V.N. Goutham, [2]Samir Kumar Panda, [3]Mrudula Vandana**

*[1*]Assistant Professor, Department of General Medicine, GIMSR, GITAM (Deemed to be University), Visakhapatnam.*
*[2]Associate Professor, Department of General Medicine, GIMSR, GITAM (Deemed to be University), Visakhapatnam.*
*[3]Assistant Professor, Department of Radio-Diagnosis, GIMSR, GITAM (Deemed to be University), Visakhapatnam.*
*Corresponding author: V.V.N. Goutham, gvalapal@gitam.edu*

## ABSTRACT

**Introduction:** Endothelial Dysfunction(ED) is produced by oxidation stress, breakdown and inactivation of nitric oxide, prostanoids, endothelin – 1 leading to decreased bioavailability of NO sustained SBP and high mean BP blunts Flow Mediated Dilatation (FMD) response in brachial artery. This particular study has been carried out to analyze brachial artery flow-mediated vasodilatation by ultrasonographic imaging with high resolution along with pulse Doppler study.

**Material and Methods:** This study has been carried out in a tertiary care hospital, Visakhapatnam during the session August 2018 to December 2019.Fifty hypertensive patients diagnosed as per JNC-VIII recommendation have been studied. Thirty males and twenty females have been taken as control for comparison. Evaluation of their endothelial function was done in terms Resting Brachial Artery Diameter (RBAD) and FMD% in hypertension group.

**Results:** In the present study serum cholesterol value in hypertension has found to be 174 ± 18.02 mg/dl, whereas, its value in control is 170.6 ± 20.5 mg/dl. HDL value found to be 47.70 ± 6.4 mg/dl in hypertension, 47.51 ± 6.50 with LVH and 48.12 ± 4.99 without LVH and in control; it is 42.6 ± 9.34 mg/dl. HDL value which is known to be protective against atherosclerosis did not show positive correlation with FMD%.

**Conclusion:** FMD% may not be useful for therapeutic purpose because large number of physiological factor alters this. It is a functional bioassay for, in vivo, endothelial function in human.

**Keywords:** Flow-mediated Dilatation, Brachial Artery Diameter, Endothelial Dysfunction, Hypertensive, FMD, Parameters.

**Correspondence:**
V.V.N. Goutham
Assistant Professor,
Department of General Medicine,
GIMSR, GITAM (Deemed to be University), Visakhapatnam.
**E-mail Address:**
gvalapal@gitam.edu

**Submitted:** 27-09-2020
**Revision:** 28-10-2020
**Accepted Date:** 26-11-2020

**DOI:** 10.31838/jcdr.2020.11.04.44

## INTRODUCTION

Endothelium is a specialized type of epithelial tissue. More specificially, it is simple squamous epithelium. Endothelium of the interior surface of the heart chambers is called endocardium. Endothelial cells release various substances which are vasoactive such as NO(nitric oxide), prostacyclin and endothelium-derived hyperpolarising factor which are helpful in regulating vascular smooth muscle and also plays an important role in trafficking of blood cells.(1) NO has antiatherogenic proprieties by preventing platelet aggregation and adhesion. It also has thromboresisitant property (2,3). Nitric Oxide is formed from L-arginine by oxidation of its guanidine-nitrogen terminal (4). In recent years, Studies published suggest that risk factors such as hypercholesterolemia, hypertension, and diabetes mellitus enhance the vulnerability of the microvasculature to the IR-induced injury responses that are mediated by adherent leukocytes. Elevated levels of plasma cholesterol exacerbate dysfunctional responses elicited in all segments of the microvasculature.

Flow-mediated dilation is referred to as an endothelium-dependent mechanism that represents an artery's relaxation when subjected to increased flow and thus increased shear stress (5). It is a diagnostic aid and also an experimental tool for endothelial dysfunction which is considered as a risk factor in coronary artery disease (CAD). Brachial Artery hemodynamic includes brachial artery diameter (D) and local blood flow velocity (V). Kinetics of changes in V&D can be determined by use of pulsed Doppler signal during reactive hyperemia.(6) During distal occlusion both velocity and diameter deceases but during hyperemia increase in flow velocity and diameter reflects degree of endothelial dysfunction.(7) FMD is also affected by high cholesterol levels, while triglyceride plasma levels do impact FMD by rioting. The impaired vasodilator response in hypercholesterolemia may result from increased NO inactivation by oxygen-free radicals produced by the presence of low-density lipoprotein cholesterol which are in oxidized form. In obese people FMD has been shown to be blunted. (8) FMD has been shown to be blunted in obese individuals. Percentage of patients with unstable anigina pectoris has concurrent diminished FMD. Plasma levels of C - reactive protein were associated with in patients with stable CAD. FMD, which confirms the relationship between inflammation and endothelium integrity, is also affected by hormonal factors such as the secretion of progesterone, oestrogen and catecholamines.

Healthy young adults with a family history of premature CAD have affected FMD, even though other risk factors were not present. This compromised brachial artery FMD not only corresponds but also correlates with a higher IMT of the common carotid artery, suggesting retrospective functional and structural improvements in vascular endothelium in the offspring of premature CAD patients.(9) Similar findings have been obtained in first-degree relatives of patients with type 2 diabetes. Endogenous factors and environmental factors, such as passive smoking, may impair flow mediated dilatation of brachial artery.

As there is no line between normal and high blood pressure, subjective thresholds have been set to identify individuals who are at elevated risk of experiencing a cardiovascular adverse event and benefit from medical treatment.

This should be focused not only on the degree of diastolic pressure, but also on age, sex, race, systolic pressure, and diseases associated with it.

For example, patients with diastolic pressure > 90 mmHg, if they undergo appropriate treatment, they are likely to have a substantial reduction in morbidity and mortality rate.

More and more, data suggest that systolic blood pressure may be more important than diastolic blood pressure, especially in those over the age of fifty. Generally if males with normal diastolic pressures but elevated systolic pressures have a cardiovascular mortality rate two and half times higher than those with similar diastolic pressures but whose systolic blood pressures are clearly normal. Age, race and sex along with other significant demographic factors influence blood pressure levels on the frequency of morbid cardiovascular events.

### Left Ventricular Hypertrophy in Hypertension

Left ventricular hypertrophy is very common which has a profound effect on cardiovascular morbidity and mortality, including stroke, myocardial infarction, congestive heart failure etc.(10) Non-invasive imaging approach has significantly improved our ability to test cardiac structural and functional features and our knowledge of LV hypertrophy's natural history. Though as a strong determinant of LV hypertrophy, hypertension is widely recognized. Blood pressure explains only a small amount of the inter-individual LV mass variability. In addition, LV hypertrophy occurs in the absence of hypertension and in some cases precedes its development. Over the past two decades, echocardiographic studies have enhanced our understanding of the etiology of hypertensive LV hypertrophy and cardiac dysfunction. Echocardiography provides visualization of structural or functional abnormalities, which appear long before clinical disease is detected. Echocardiographically measured LV mass has been validated and has a considerable accuracy.(11) The use of echocardiography to detect irregularities of LV diastolic filling and systemic arterial compliance has also made progress. Such studies have collectively shown the high incidence and prevalence of LV hypertrophy, cardiac dysfunction and irregular arterial compliance in patients with hypertension. However, the heart's response to hypertension is considerably inter-individual variable. Some individuals develop LV hypertrophy at equivalent blood pressure levels, while others do not, suggesting a hereditary suspectibility of LV hypertrophy development. Because of the cardiovascular manifestations of hypertension and not the blood pressure level per se, the key triggers in the hypertensive patients of morbidity and mortality remain an intense concern for understanding the genetic vulnerability of LV hypertrophy to hypertension.

Clinically, endothelial dysfunction in hypertension has an opposite effect not only in vascular tone, but also inhibits and activates such mechanisms as platelet aggregation, the proliferation and migration of vascular, smooth, cellular cell muscles, monocyte adhesion and molecular expression of adhesion that exerts at an important root level. This link between endothelial dysfunction and atherosclerosis has led to the suggestion that an important goal is to combat endothelial-dependent vasodilatation in medical treatment of hypertension.(12)

## MATERIALS AND METHODS

The present study has been undertaken in a tertiary care hospital, Visakhapatnam, Andhra Pradesh during the period of August 2018 to December 2019. Material of this study constitute of 50 healthy subjects taken as control and 50 patients of hypertension. Patients who are diagnosed to have hypertension basing on the following criteria laid down in Eighth joint national committee, were taken. After obtaining informed consent as per ethical committee recommendations selected cases were admitted to medical wards. Detailed history as per proforma, through clinical examination supported by necessary laboratory tests and imaging studies were carried out as follows: Bio-chemical tests like – Lipid profile (TC, HDL), BMI, others like – Chest X-Ray P/A View, ECG, fundoscopic examination, CBC and other routine examinations were done. In both control and study group, endothelial dysfunction was evaluated by measurement of brachial artery flow mediated dilatation. Exclusion criteria Patients of diabetes mellitus, morbid obesity, coronary artery disease (CAD), cerobrovascular accidents (CVA), subarachnoid hemorrhage (SAH), and hypertensive emergensies are excluded from the study.

Since hypertension is associated with multi organ dysfunction and cardiovascular morbidity and mortality, the present study is undertaken:

(i) To evaluate endothelial dependant vasodilatation in brachial artery and

(ii) To Co-relate endothelial dependant vasodilatation with clinical profile of patients of hypertension.

Ethical committee clearance and informed consent from each patient was taken, patient was admitted to indoor beds and was instructed to abstain from alcohol, smoking caffeine and food for eight hours. He was asked to lie quietly supine position for ten minutes. Then resting or base line diameter of right brachial artery was measured by B-Mode high resolution USG Doppler. Brachial artery was scanned in longitudinal section by placing the transducer in antecubital fossa, three centimeter above the elbow. After basal recording, forearm ischemia was induced by inflating the pneumatic cuff and following reactivate hyperemia again brachial artery diameter was measured.

In addition to blood pressure recording, ECG and biochemical parameter required for diagnosis and inclusion of cases, special tests for endothelial dysfunction includes high resolution USG, pulse Doppler system.

All data was collected from control and study group consisting of lipid profile, BMI, SBP and DBP and brachial artery diameters were analyzed in tabular form and correlated. To test if there is any significant difference of FMD% between case and control groups.

## RESULTS

The below tables shows distribution of cases according to sex. The number of males in the age group of 40 – 50, 51 – 60 and 61 – 70 are 26%, 12% and 22% respectively. The corresponding number of females being 20%, 8% and 12% respectively.

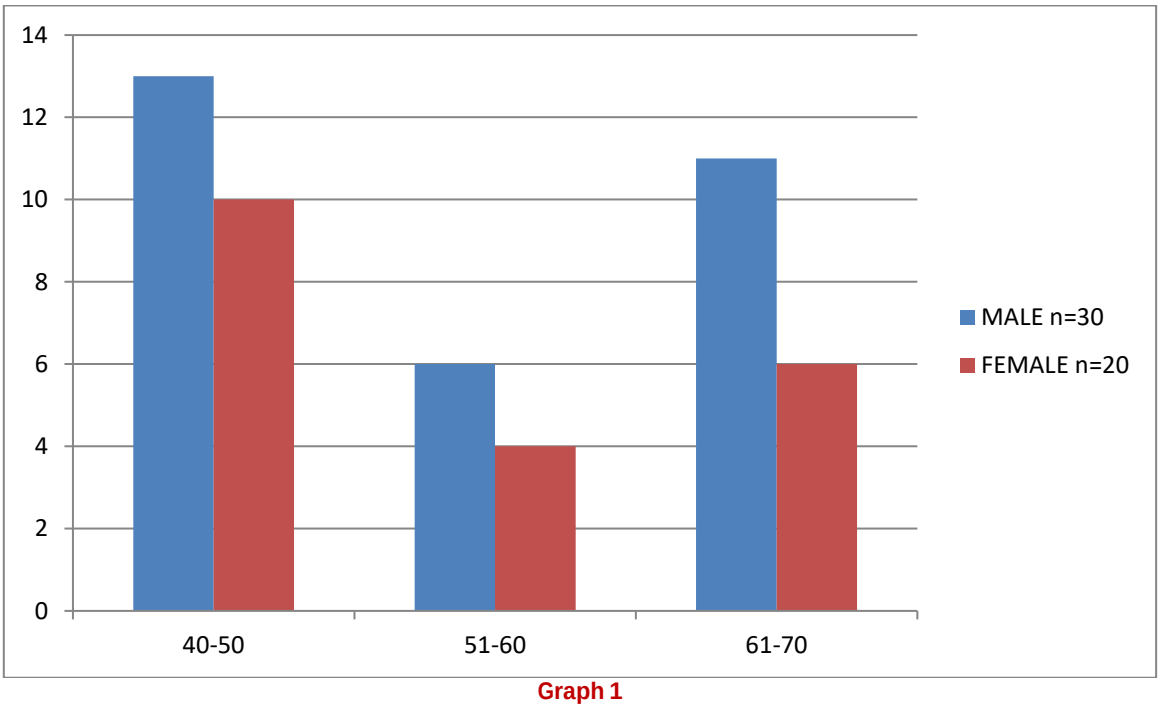
**Table 1**

| Distrubition Of Cases According To Sex (N=50) | | | | |
|---|---|---|---|---|
| Age | Male (N=30) | % | Female (N=20) | % |
| 40-50 | 13 | 26 | 10 | 20 |
| 51-60 | 6 | 12 | 4 | 8 |
| 61-70 | 11 | 22 | 6 | 12 |
| TOTAL | 30 | 60 | 20 | 40 |

**Table 2**

| Distrubition Of Controls According To Sex (N=50) | | | | |
|---|---|---|---|---|
| Age | Male (N=28) | % | Female (N=22) | % |
| 40-50 | 20 | 40 | 11 | 22 |
| 51-60 | 7 | 14 | 10 | 20 |
| 61-70 | 1 | 2 | 1 | 2 |
| TOTAL | 28 | 56 | 22 | 44 |

The above table shows distribution of controls according to sex. The number of males in the age group of 40 – 50, 51 – 60 and 61 – 70 are 40%, 14% and 2% respectively. The corresponding number of females being 22%, 20% and 2% respectively.




**Graph 1**

**Table 3**

| Relationship of total cholestrol and fmd% in control group | | |
|---|---|---|
| Total cholestrol (mg/dl) | No. Of Controls | Fmd (%) Mean sd |
| 140-150 | 1 | 11.50 |
| 151-160 | 11 | 12.88 |
| 161-170 | 14 | 12.25 |
| 171-180 | 13 | 12.82 |
| 181-190 | 11 | 12.61 |

The above table shows relationship of serum cholesterol value with FMD%.

**Table 4**

| Relationship of total cholestrol and fmd% in hypertensive (cases) | | |
|---|---|---|
| Total cholestrol (mg/dl) | No. Of cases | Fmd (%) mean ± sd |
| 151-160 | 6 | 6.50 |
| 161-170 | 8 | 8.05 |
| 171-180 | 14 | 5.47 |
| 181-190 | 18 | 5.46 |
| 191-200 | 4 | 5.48 |

The above table shows correlation of FMD% with total cholesterol value in hypertensive group as decrease in FMD% above 170 mg percentage.

**Table 5**

| Study sample characteristic | | |
|---|---|---|
| | Normal | Hypertension |
| Age | 56±14 | 55±15 |
| M:F | 28.22 | 30:20 |
| Total cholesterol (mg/dl) | 170.6±20.5 | 174±18.2 |
| HDL (mg/dl) | 42.26±9.34 | 47.70±6.04 |
| B.M.I (Kg/m2) | 23.4±3.2 | 21.6±3.4 |
| SBP (mmHg) | 120±10.4 | 170.4±14.8 |
| DBP (mmHg) | 74±12 | 110±8.2 |
| RBAD (mm) | 3.97±0.61 | 4.10±0.5 |
| HBAD (mm) | 4.30±0.94 | 4.42±0.54 |

The above table shows comparison of different clinical and biochemical parameters between control and study group.

**Table 6**

| Study sample characteristic | | |
|---|---|---|
| | Hypertension without LVH | Hypertension with LVH |
| Age | 49.57±6.37 | 65.13±4.42 |
| M:F | 21:14 | 9:6 |
| Total cholesterol (mg/dl) | 175.91±11.10 | 187.07±10.52 |
| HDL (mg/dl) | 48.12±4.99 | 47.51±6.50 |
| B.M.I (Kg/m2) | 23.77±2.35 | 25.22±2.12 |
| SBP (mmHg) | 159±10.2 | 172±14.4 |
| DBP (mmHg) | 104±3.4 | 110±4.8 |
| RBAD (mm) | 3.98±0.54 | 4.06±0.46 |
| HBAD (mm) | 4.21±0.78 | 4.28±0.47 |

The above table shows comparison of clinical and biochemical parameters in the study group of having LVH and without LVH.

**Table 7**

| Brachial artery FMD model | |
|---|---|
| | FMD% |
| Normal (n=50) | 12.4±2.2 |
| Hypertension (n=50) | 5.6±2.4 |
| Hypertension without LVH (n=35) | 6.8±3.6 |
| Hypertension with LVH (n=15) | 3.4±1.8 |

The above table shows correlation of FMD% between normal, study group and study group having hypertension without LVH and with LVH.

### Analysis the Relationship between the Blood Pressure and FMD%

Number of females being 11(22%), 10(20%) and 1(2%) respectively. Distribution of FMD% in control group having SBP from 101-110, 111-120 and 121-130 are 13.57%, 12.49% and 12.58% respectively and distribution of FMD% in cases group having SBP from 131-141, 141-150, 151-160, 161-170, 171-180 and 181-190 are 6.15%, 6.74%, 5.56%, 5.62%, 5.02% respectively. Distribution of FMD% in controls having diastolic blood pressure ranges from 60-70, 71-80 and 81-90 are 12.06%, 12.76% and 12.55% respectively and distribution of FMD% in cases having diastolic blood pressure ranges from 91-100, 101-110 and 111-120 are 5.77%, 6.19% and 3.10% respectively.

## DISCUSSION

Endothelial dysfunction has been evaluated by using high resolution ultrasonography with Doppler system to observe on resting brachial artery diameter and change in its diameter after induction of ischemia by forum occlusion. In addition to calculation of FMD% various parameters connected to blood flow and dilatation in brachial artery has been collected.(13)

In this study of hypertension patient of age group of 40 - 70 have been included, 26% of male and 20% of female are in 4$^{th}$ decade and male preponderance (60%) is observed.

Observation on hypertensive patients over a wide range of SBP from 131 - 190 mmHg shows gradual decline in FMD% with increase of SBP. In 151 - 160 mmHg, SBP range, FMD% was 6.74%, whereas in 181 - 190 mmHg SBP range it significantly decreases to 5.02%.

Similarly, diastolic blood pressure range increasing from 91 - 100 mmHg to 111 - 120 mmHg shows proportional decrease in FMD% from 5.77% to 3.10%.

In present study reveals that with abnormal lipid profile and high serum cholesterol value FMD% is blunted. Serum cholesterol value in hypertension has found to be 174 ± 18.02 mg/dl, whereas, its value in control is 170.6 ± 20.5 mg/dl. HDL value found to be 47.70 ± 6.4 mg/dl in hypertension, 47.51 ± 6.50 with LVH and 48.12 ± 4.99 without LVH and in control; it is 42.6 ± 9.34 mg/dl. HDL value which is known to be protective against atherosclerosis did not show positive correlation with FMD%.(14,15)

The mechanism of involved in atherosclerosis is multi-factorial and is known to be aggravated by hypercholesterolemia. Early athermanous vascular changes, the so called fatty streak lesion persistent in adolescent. (16)

BMI has been found to be 23.4 ± 3.2 kg.m2 in control and 21.6 ± 3.4 kg/m$^2$ in hypertension. In LVH group the value is 25.22 ± 2.12 kg/m$^2$ whereas, in non LVH group it is 23.7 ± 2.35 kg/m$^2$.(17)

Though, serve obesity with BMI >30 have been excluded from this study, the available data shows that in hypertension with LVH group where BMI is 25.22 ± 2.12 kg/m2 FMD% is lowest i.e., 3.4 ± 1.8%.

Among the factors impending FMD%, obesity is one. The mechanism of obesity induced endothelial dysfunction appears to be multi-factorial including dyslipidemia, elevated blood pressure, increasing inflammation, oxidative stress, and rate of glucose metabolism. Additionally, low calorie diet, carbohydrate restriction excessive, and change in plasma glucose. Concentration improve FMD% and enhance endothelial dependent vasodilatation.

In present study, FMD% has been evaluated in four groups like control (n=50), hypertensive (n=50), with LVH (n=15) and hypertensive without LVH (n=35).

FMD% in normal and hypertensive groups are 12.4 ± 2.2% and 5.6 ± 2.4% respectively. Whereas, FMD% in hypertension without LVH and with LVH are 6.8 ± 3.6% and 3.4 ± 1.8% respectively.(18)

FMD% value in control and hypertensive group shows statistically significant decrease (pvalue = <.001)

Takeshi Moatayama et al; have reported 8.0 ± 2.5% in control Vs 5.6 ± 3.0 in hypertensive group. Their report value in LVH group and non LVH group are 2.9 ± 2.6% and 4.2 ± 1.8% respectively.

Higher values of FMD% encountered in present study may be explained by several factors influencing FMD% measurement. Important factors which can be altered are:

- Time of test - 7 A.M to 9 A.M., fasting, abstinence from caffeine, smoking etc.
- Site of cuff - distal or proximal, i.e., either 5 cm above or below elbow.
- Exact determination of site of transducer (at an angel of 70$^o$ to center of artery.

- Proper identification of center of blood column in brachial artery.
- Pressure and duration of cuff inflation
- After release of cuff, scanning is started at 60 seconds. Alternately, the first scan is taken at 45 seconds, 60 seconds, 90 seconds or continuous scan from 30 seconds before release to 90 seconds after deflation of cuff.
- Such continuous scanning over a prolonged period would be highly informative for flow velocity accuracy.
- Transducer should be maintained in a fixed position without shaking in relation to patient's arm.

Since measurement of brachial artery diameter is procedure dependent, observe dependent and is influenced by all above parameters, variation in FMD% projection by various study groups may be valid.(19,20)

Significant decrease of FMD% in hypertensive group compare to control (5.6 ± 2.4% Vs 12.4 ± 2.2%) reflects a series of events following forearm occlusion and pathophysiologic sequence occurring in brachial artery and its wall differently in both groups.(21)

While comparing the observation in two separate groups like:

Hypertension with LVH (n) = 15
Hypertension without LVH (n) = 35

### Hypertensive with LVH Group

| | | |
|---|---|---|
| Hypertension with LVH group | = | 65.13 ± 4.42 years |
| Male: Female | = | 9.6 (male preponderance) |
| Total cholesterol | = | 187.07 ± 10.52 mg / dl, |
| HDL | = | 47.51 ± 6.50mg/dl |
| BMI | = | 25.22 ± 2.12 kg/m2 |
| SBP | = | 172 ± 14.4 mmHg, |
| DBP | = | 110 ± 4.8 mmHg |
| RBAD | = | 4.06 ± 0.46 mm, |
| HBAD | = | 4.28 ± 0.47 mmHg |
| FMD% | = | 3.4 ± 1.8% |

### Hypertensive without LVH Group

| | | |
|---|---|---|
| Hypertension with LVH group | = | 49.57 ± 6.37 years |
| Male: Female | = | 21.14 (male preponderance) |
| Total Cholesterol | = | 175.91 ± 11.10 mg / dl, |
| HDL | = | 48.12 ± 4.99 mg/dl |
| BMI | = | 23.77 ± 2.35 kg/m2 |
| SBP | = | 159 ± 10.2 mmHg, |
| DBP | = | 104 ± 3.4 mmHg |
| RBAD | = | 3.98 ± 0.54 mm, |
| HBAD | = | 4.21 ± 0.78 mmHg |
| FMD% | = | 6.8 ± 3.6% |

## CONCLUSION

The data comparing both group of hypertension reflects that the group with LVH is associated with increased risk factors like hypercholesterolemia, aging, increased BMI and pattern of blood pressure which is higher and sustained sp as to produce ECG positive LVH. Impaired FMD% can be related to old age, sex, OCP, insulin, resistance, obesity and hypertension. Reactive hyperemia is the stimulus for FMD%.

It can be postulated that described hyperemia leads to decrease stimulus. Shear stress computed as area under the curve contributes significantly to FMD%. The proposed mechanisms for FMD are 1) Superoxide mediated inactivation of NO 2)Exercise induced up regulation of eNOs 3)Endothelium derived Prostanoids (can be blocked by Indomethacin) 4)Hypoxia of smooth muscles 5)Altered myogenic Response. FMD% is a diagnostic aid for evaluation of endothelial function. (22)

## SOURCES OF SUPPORT

Nil

## CONFLICTS OF INTEREST

None

## REFERENCES

1. Palmer RMJ, Ferige AG, Moncada S. Nitric Oxide releases accounts for the biological activity of endothelium-derived relaxing factor. *Nature* 1987; 327(6122): 524-526.
2. Joannides R, Haefeli WE, Linder L, Richard V, Bakkali EH, Thuillez C, Lüscher TF. Nitric oxide is responsible for flow-dependent dilatation of human peripheral conduit arteries in vivo. *Circulation* 1995; 91(5): 1314-1319.
3. Fukai T, Siegfried MR, Ushio-Fukai M, Cheng Y, Kojda G, Harrison DG. Regulation of the vascular extracellular superoxide disunitase by nitric oxide and excersice training. *The Journal of clinical investigation* 2000; 105(11): 1631-1639.
4. Adams MR, McCredie R, Jessup W, Robinson J, Sullivan D, Celermajer DS. Oral L-arginine improves endothelium-dependent dilatation and reduces monocyte adhesion to endothelial cells in young men with coronary artery disease. *Atherosclerosis* 1997; 129(2): 261-269.
5. Laurent S, Lacolley P, Brunel P, Laloux B, Pannier B, Safar M. Flow-dependent vasodilation of brachial artery in essential hypertension. *American Journal of Physiology-Heart and Circulatory Physiology* 1990; 258(4): H1004-H1011.
6. Anderson EA, Mark AL. Flow-mediated and reflex changes in large peripheral artery tone in humans. *Circulation* 1989; 79(1): 93-100.
7. Levenson JA, Peronneau PA, Simon A, Safar ME. Pulsed Doppler: determination of diameter, blood flow velocity, and volumic flow of brachial artery in man. *Cardiovascular Research* 1981; 15(3): 164-170.
8. Clarkson P, Celermajer DS, Donald AE, Sampson M, Sorensen KE, Adams M, Deanfield JE. Impaired vascular reactivity in insulin-dependent diabetes mellitus is related to disease duration and low density lipoprotein cholesterol levels. *Journal of the American College of Cardiology* 1996; 28(3): 573-579.
9. Neunteufl T, Katzenschlager R, Hassan A, Klaar U, Schwarzacher S, Glogar D, Weidinger F. Systemic

endothelial dysfunction is related to the extent and severity of coronary artery disease. *Atherosclerosis* 1997; 129(1): 111-118.
10. Casale PN, Devereux RB, Milner M, Zullo G, Harshfield GA, Pickering TG, Laragh JH. Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. *Annals of internal medicine* 1986; 105(2): 173-178.
11. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *New England Journal of Medicine,* 1990; 322(22): 1561-1566.
12. Iiyama K, Nagano M, Yo Y, Nagano N, Kamide K, Higaki J, Ogihara T. Impaired endothelial function with essential hypertension assessed by ultrasonography. *American heart journal* 1996; 132(4): 779-782.
13. Bae JH. Noninvasive evaluation of endothelial function. *Journal of cardiology* 2001; 37(1): 89-92.
14. Drexler H, Zeiher AM. Endothelial function in human coronary arteries in vivo. Focus on hypercholesterolemia. *Hypertension* 1991; 18(4): 1190-1199.
15. Mullen MJ, Kharbanda RK, Cross J, Donald AE, Taylor M, Vallance P, Mac Allister RJ. Heterogenous nature of flow-mediated dilatation in human conduit arteries in vivo: relevance to endothelial dysfunction in hypercholesterolemia. *Circulation research* 2001; 88(2): 145-151.
16. Ross R. The pathogenesis of atherosclerosis—an update. *New England journal of medicine* 1986; 314(8): 488-500.
17. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Annals of internal medicine* 1991; 114(5): 345-352.
18. Fan L, Santago P, Jiang H, Herrington DM. Ultrasound measurement of brachial flow-mediated vasodilator response. *IEEE transactions on medical imaging* 2000; 19(6): 621-631.
19. Corretti MC, Anderson TJ, Benjamin EJ, Celermajer D, Charbonneau F, Creager MA, Vallance P. Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: a report of the International Brachial Artery Reactivity Task Force. *Journal of the American College of Cardiology* 2002; 39(2): 257-265.
20. Corretti MC, Plotnick GD, Vogel RA. Technical aspects of evaluating brachial artery vasodilatation using high-frequency ultrasound. *American Journal of Physiology-Heart and Circulatory Physiology* 1995; 268(4): H1397-H1404.
21. Ghiadoni L, Huang Y, Magagna A, Buralli S, Taddei S, Salvetti A. Effect of acute blood pressure reduction on endothelial function in the brachial artery of patients with essential hypertension. *Journal of hypertension* 2001; 19(3): 547-551.
22. Schroeder S, Enderle MD, Ossen R, Meisner C, Baumbach A, Pfohl M, Karsch KR. Noninvasive determination of endothelium-mediated vasodilation as a screening test for coronary artery disease: pilot study to assess the predictive value in comparison with angina pectoris, exercise electrocardiography, and myocardial perfusion imaging. *American heart journal* 1999; 138(4): 731-739.

**Cite this article:** V.V.N. Goutham Relationship Analysis between Flow Mediated Dilatation of Brachial Artery and Hypertension along with Other Related Parameters. J. Cardiovascular Disease Res., 2020; 11 (4): 249 – 254