

An ascending aortic haemodynamic comparison between normotensive and hypertensive subjects

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ABSTRACT

Background: Systemic hypertension, a common disorder with potentially serious complications, exerts ill effects through structural and functional modifications of arterial wall. Haemodynamics play an important role in the development of atherosclerosis. Local hemodynamic temporal pressure and wall shear stress are important for understanding the mechanisms leading to various complications in cardiovascular function.

Objectives: Since we could not find such a study in literature involving Indian population, this prompted us to investigate and establish the relationship between the blood pressure and the ascending aortic pulse wave parameters in normal individuals and compare the same with hypertensives.

Material and methods: A case control study was done in a tertiary care hospital involving 25 hypertensive patients and further compared with 25 normotensive subjects of same age group acting as control. The GE-P 100 Doppler echocardiography machine was used to study acceleration time, deceleration time, ejection time, ejection fraction, peak flow velocity and pressure gradient in hypertensives and compared the same with age matched normotensive. Also ascending aortic diameter was mapped at the annulus.

Results: The results of our study confirmed our assumption that in hypertensive the ascending aortic haemodynamic parameters are abnormal and both systolic and diastolic blood pressure does exert a statistically significant influence on the Doppler parameters of ascending aorta. The acceleration time, deceleration time, pressure gradient and ejection time showed statistically significant increase in hypertensives when compared to normotensive. At the same time the ejection fraction and the ascending aortic diameter showed a statistically significant decrease than normotensive.

Conclusion: We would conclude to say that the Doppler parameters of ascending aortic blood flow are abnormal in hypertensives. This altered haemodynamics may lead to further ill effects by way of altered peripheral haemodynamics. This Doppler evaluation of ascending aortic blood flow can be developed as a clinical tool for evaluating hypertensives and assessing the benefit of treatment of hypertension.

Key words: Ascending Aorta, haemodynamics, acceleration time, deceleration time, hypertensives

Introduction

The role of the heart is to provide body tissues with a continuous stream of blood. The heart fulfils this function by converting potential energy into kinetic energy. The heart should normally be able to pump blood against pressures upto 300 mm Hg and the flow must not exceed the limit of tolerance for mechanical trauma to blood vessels and the blood corpuscles.

Aorta functions not only as a conduit delivering blood to tissues but also as an important modulator of entire cardiovascular system. The viscoelastic and compliant properties of the aorta also sub serves the buffering of intermittent pulsatile output from left ventricle, to provide steady flow to capillary beds. Because of continuous exposure of ascending aorta to high pulsatile pressure

and shear stress, it is prone to injury and disease, resulting from mechanical trauma altering the normal physiology of aortic function. One such cause exposing the ascending aorta to excessive mechanical trauma and altering the physiology of aortic blood flow is systemic hypertension.

The blood column in the root of aorta is rapidly accelerated and pushed towards the periphery during the rapid ejection phase and the flow slows during the reduced ejection phase. There is histological evidence that in aortic wall, in muscle cells, elastic fibres and some collagenous fibres are interlinked into a three dimensional network. With more loads, these elastic fibres could become stiffer and other components of the network could also be increasingly involved.^[1,2]

Systemic hypertension, a common disorder with potentially serious complications exerts ill effects through structural and functional modifications of arterial wall. Increased wall stress stimulates cellular changes in the structure i.e., cell elongation in the direction of stress.^[3]

Haemodynamics play an important role in the development of atherosclerosis. Low flow, low wall shear stress have been hypothesized to play a role in atherosclerotic lesion locations. Haemodynamics is concerned with the forces generated by the heart and the resulting motion of blood through the cardiovascular system. The haemodynamics of ascending aorta is determined by a number of factors, some of them are:

- Systemic arterial blood pressure
- Pressure gradient across its length

- Its dimensions
- Shear stress on the vessel wall
- Left ventricular output
- Properties of blood
- Age

Previous studies using different techniques have shown that with increased arterial blood pressure, haemodynamics of ascending aorta may be altered. This may be due to an increase in peripheral resistance or by changes in peripheral arterial walls^[4-7], or by degeneration of its normal components.^[8]

A study describing the mechanism of altered haemodynamics revealed that the flow in ascending aorta is disturbed but not turbulent. The mechanism responsible for this disturbance was postulated to be that during systole the blood ejected into the aorta separates from a slower moving boundary layer near the walls and at the edges of the valve cusps.^[9]

The proximal vessels have to convert cardiac pulsations into a more constant flow pattern in the distal circulation for which the proximal vessels should have a good compliance, which in turn is dependent on specific structural elements within the arterial wall especially muscle, elastic and collagen fibres. Hypertension has been found to affect the large elastic arteries more which in turn may contribute to circulatory physiology alterations. One of the hallmarks in hypertension is an inappropriate degree of vasoconstriction for any given level of flow. This haemodynamic abnormality is an example of dynamic cardiovascular disregulation.^[10]

The chronic hypertension has been described as a condition in which the sympathetic nervous system and renin

angiotensin system continue to exert chronic pressor effects on heart and vasculature. It has been further emphasized that the hypertension-induced alterations in the vasculature of aorta is a vicious cycle playing through an integrated system of control.^[11]

Hypertension has long been considered a haemodynamic disorder. Shear force, one of the vital mechanical factors contributing to changes in arterial structure is now an important field for clinical research. In hypertension, the sub endothelial layers of vessels tend to degenerate locally; one reason is the high velocity of blood flow associated with the shear stress. Shear stress is the product of the shear rate and viscosity.^[12]

With the advent of non invasive measurement of haemodynamics, it is now possible for continuous monitoring of haemodynamics not only in hypertensives but in various other cardiovascular disorders as well as renal failures.

Direct evidence was obtained by measuring carotid – femoral pulse wave velocity, that the aortic stiffness is an independent predictor of all cause and cardiovascular mortality in patients with essential hypertension.^[13]

Systemic haemodynamics, is the sum of interactive forces of pulmonary vasculature, the left heart and systemic vasculature. To a clinician, these forces demonstrate themselves as a pressure-flow relationship at the output mode of the left heart.

Arterial pulse waves generated by the force of each contraction are both the pressure wave and the associated velocity wave. The best and most widely used technique to estimate the distensibility and stiffness of aorta is pulse wave velocity.

The Doppler aortic flow velocity measurements can be used to assess quantitatively global left ventricular performance at rest and after pharmacological and other haemodynamic interventions.

The recent interest in ascending aortic haemodynamics is due to the fact that a significant majority of cardiovascular disorders like essential hypertension is related to systemic haemodynamics. With the validation of a third generation Doppler velocity system for studies of detailed aortic haemodynamics^[14] it has become easier to evaluate the ascending aortic physiological function.

There are various reports available on Doppler haemodynamic study of ascending aorta in various age groups in normal individuals and also in hypertension patients. But due to lack of similar studies involving Indian subjects this study was undertaken. The purpose of this study is to evaluate the ascending aortic haemodynamics in the hypertensives and compare them with the normotensive individuals.

Material and methods

Various studies till date have shown that sustained hypertension caused increased diameter and slight decrease in velocity of blood flow in the arteries. This study is based on the hypothesis, that “DOPPLER AORTIC VELOCITY PARAMETERS ARE ABNORMAL IN HYPERTENSIVES WHEN COMPARED TO THAT OF NORMOTENSIVES”

Aortic haemodynamic effects of hypertension have not been much investigated so far, to prove or disprove this hypothesis. This study was designed with the aim to test the current

hypothesis vis-à-vis assess the effect of hypertension on ascending aortic haemodynamics.

Source of data

This is a case control study and hence 25 cases of hypertensive patients attending cardiology department at M.S. Ramaiah Medical College & Teaching Hospital were studied and further compared with 25 normotensive subjects of same age group acting as controls.

Method of collection of data

Twenty-five essential hypertensive patients attending cardiology department at M.S. Ramaiah Medical College & Teaching Hospital who satisfy the inclusion criteria were subjects for this study. This study was conducted from Aug 2002 to Dec 2003. JNC VI, 1992 guidelines for hypertension was followed.

INCLUSION CRITERIA

- Essential Hypertensive Patients.
- Patients between 30 to 50 years of age, of both sexes.
- On drug therapy whether controlled or uncontrolled.
- Of more than three months duration
- With or without left ventricle hypertrophy

EXCLUSION CRITERIA

- Patients with aortic valve disease (Ruled out by Echocardiography)
- Patients with any other cardiac illness (Ruled out by ECG and Echocardiography)
- Patients with diabetes (Ruled out by Glucose Random Blood Sugar)
- The above said 25 hypertensive patients were compared with age controlled 25 normotensive subjects, who were volunteers.

SYSTEM DESIGN

The GE-P 100 Doppler echocardiography machine was used for the study. The transducer for ascending aortic flow recordings was of low frequency. This system allowed the location of the Doppler sample volume to be superimposed on a two-dimensional image of heart so that the sample volume could be placed within the ascending aorta parallel to the long axis of the blood stream.

The sector scanner used in the present study was equipped with a 3 Mhz transducer with a half power beam width of approximately 3 mm transducer with a half power beam width of approximately 3 mm at the sample depths utilized to record blood flow velocity in the ascending aorta.

The Doppler frequency shifts were processed by a dual-channel, real time spectrum analyser and displayed at 5 msec intervals on a strip chart recorder. Doppler flow recordings were displayed as blood flow velocity in cms / sec vs. time. Stop frame hard copy images of Doppler M mode and 2 DE data were recorded. The frozen images were recorded on a glossy black-on-white electrostatic paper at a speed of 50 mm/sec.

PROTOCOL DESIGN

Informed consent was obtained from the volunteers and the hypertensive patients visiting cardiology department. Ethical clearance was obtained from the institutional ethics committee. Aortic spectral Doppler was recorded in the left lateral position in apical area, in held respiration by means of standard techniques. Ascending aortic blood flow velocity was measured by placing a right angle, 3 MHz M-mode transducer in the

apical view using pulsed Doppler approach for the 3 MHz transducer.

The range velocity was approximately 1350 cm² which permitted peak flow velocities upto 170 cm/sec at a range of 8 cm. The flow signal used for analysis was that flow signal which was characterized by the greatest maximum blood flow velocity. The ultrasonic beam was kept parallel to the moving column of blood in the ascending aorta. The waveform with the greatest amplitude is selected for a detailed analysis.

An attempt was made to minimize the effect of respiration by choosing the beat showing greatest peak flow. When the greatest peak flow velocity was seen on the screen, the image was stop framed and recorded. After completing the Doppler recording the 2-d imaging was turned on to confirm the position of sample volume in the ascending aorta.

Aortic flow measurements made from non imaging technique recordings gives a number of flow parameters including peak flow velocity ejection time, acceleration time and flow velocity integral have been measured. [9] Five routine echo views were taken:

- Left parasternal view
- Apical two chamber view
- Four chamber view
- Long axis view
- Short axis view

Additionally suprasternal view was also obtained for the said parameter. An average of three values was obtained.

STATISTICAL ANALYSIS

Data were stored and analysed by the SPSS 11.0 statistical package. Mean values are presented as index of dispersion. Differences between mean

values of hypertensives and normotensive were tested for statistical significance with Independent Student t test. Statistical significance of the hemodynamic Doppler parameters measured was evaluated using paired student t test. A value of P<0.05 was considered statistically significant*, P<0.02 was considered statistically highly significant ** and P<0.01 was considered statistically very highly significant. ***

Results

The demographic data of normotensive and hypertensives included in the study were recorded. The age, height, weight, body surface area, packed cell volume between normotensive and hypertensives were compared. (Table 1) The mean age of normotensive was 41 years (6.45) and height 168 cms (2.71) were compared with hypertensives of age and height being 44.2 years (6.45) and height 169.75 cms (2.71) respectively. When the mean acceleration time (89.5 m/s), deceleration time (207.2 m/s), pressure gradient (4.73mm/Hg) and ejection time (287.9 m/s) were compared with the hypertensives, the values showed statistically significant increase with the mean acceleration time being 125.04 m/s, deceleration time being 233.12 m/s, pressure gradient being 5.55 mm/Hg and ejection time of 339 m/s in age and height matched hypertensives. At the same time when the mean ejection fraction (63.3 %) and the ascending aortic diameter (1.533 cms) were compared with the hypertensives they showed a decrease which was statistically significant with ejection time being 53.06 % and ascending aortic diameter being 1.73 cms. (Table 2)

Table 1: Comparison of age, height, weight, body surface area, packed cell volume between normotensive and hypertensives

	NORMOTENSIVES				HYPERTENSIVES			
	MIN	MAX	MEAN	SD	MIN	MAX	MEAN	SD
AGE (YRS)	30	53	41	6.455	31	60	44.2	6.677
HEIGHT(CMS)	163	173	168	2.71	165	174	169.75	2.65
WEIGHT (Kg)	52	70	59.44	4	52	80	60.76	6.25
BSA(Sq Mt)	1.97	2.12	1.99	0.011	1.85	2.13	1.97	0.004
PCV (%)	37	44	40.56	2.21	37	44	40.7	2.758
SBP	106	120	114.72	4.99	120	160	144	11.04
DBP	74	86	79.76	2.9	90	104	94.72	4.11

Table 2: Comparison of Doppler ascending aortic hemodynamic parameters between normotensive and hypertensives

	NORMOTENSIVES			HYPERTENSIVES			STUDENT'S t TEST
	MIN	MAX	MEAN	MIN	MAX	MEAN	P VALUE
ACCT	77	112	89.5	95	192	125.04	0***
DECCT	168	240	207.2	160	276	233.12	0.001**
ET	260	332	287.9	238	391	339	0***
PFV	88	131	113.12	88	138	118	0.212
PG	3.35	5.79	4.73	2.75	7.3	5.55	0.001**
EF	43.42	80	63.3	31.74	72.16	53.06	0.001**

ACCT-Acceleration time in m/sec
 DECCT-Deceleration time in m/sec
 PFV- Peak flow velocity in mm/Hg
 PG- Pressure gradient in mm/Hg
 ET- Ejection Time
 EF- Ejection Fraction

Discussion

On the basis of stretch curves, it is proved that the elastic tissue is much more extensible than collagenous fibres in hypertension. When a wave encounters a boundary which is neither rigid (hard) nor free (soft) but instead somewhere in

between, part of the wave is reflected from the boundary and part of the wave is transmitted across the boundary. The exact behaviour of reflection and transmission depends on the material properties on both sides of the boundary. This cause's characteristic impedance in

ascending aorta which in turn disturbs the normal relationship between components of left ventricular ejection wave and impedance presented to the left ventricle and results in increased pulsatile energy losses in the vascular system. The decreased arterial distensibility in hypertensives may be due to changes in arterial wall elastic components itself.^[9]

Further in our study, we evaluated the effect of decreased ascending aortic diameter on the pressure - flow waves recorded using Doppler echocardiograph and compared the same with normotensive individuals. At the ascending aorta Flow is inertio-elasatic. Elasticity is promoted by the blood and vessel wall structure.^[4]

We found a very highly statistically significant increase in the acceleration time and deceleration time of the pulse wave in hypertensives. This probably could be due to increased resistance offered by the decreased diameter at the annulus of ascending aorta.

The peak flow velocity in hypertensives was found to have a little increase compared to normotensive individuals but was statistically not significant. This probably may be due the increased stiffness and decreased distensibility of the vessel causing a reduction in the ejectile velocity of blood flow through the blood pumped from left ventricle is of much greater force in hypertensives.^[15]

The pressure gradient across ascending aorta showed a very highly significant increase in hypertensive subjects compared to normotensive individuals. This is obvious as the decreased opening angle, increased stiffness and decreased distensibility of aorta results in a greater increase in the

pressure in the left ventricle thereby causing an increase in pressure gradient in hypertensives.^[16]

We also found a very high statistical significant increase in the ejection time in hypertensive patients. This again can be attributed to decreased opening angle, increased stiffness and decreased distensibility of aorta. Increased stiffness of aortic wall causing an increased aortic pressure leads to increased left ventricular ejection time, which in turn leads to an increased duration for which the aortic valves are kept open.^[16]

We would conclude to say that the Doppler parameters of ascending aortic blood flow are abnormal in hypertensives. This altered haemodynamics may lead to further ill effects by way of altered peripheral haemodynamics. Hypertension is one of the common cardiovascular diseases. This Doppler evaluation of ascending aortic blood flow can be developed as a clinical tool for evaluating hypertensives and assessing the benefit of treatment of hypertension. This may help in achieving a reduction in morbidity and mortality due to hypertension induced cardiovascular diseases. We need to conduct the study involving large number of normotensive to get the normative ascending aortic haemodynamics data. A limitation of this study was that hypertensives duration of hypertension was not taken into consideration.

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