International Journal of Biomedical Research

ISSN: 0976-9633 (Online)
Journal DOI:10.7439/ijbr
CODEN:IJBRFA

Research Article

Functional arterial stiffness: The correlation between pulse volume and age in apparently healthy subjects

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Abstract

Background: Arterial wall thickness occurs with age in humans and is a strong predictor of cardiovascular disease risk. Age-related increase in arterial wall thickness is observed in the absence of atherosclerosis and hypertension. Measures of wall thickness are currently used as surrogates for and intermediate end points of atherosclerosis. Pulse volume refers to the movement of the vessel wall during the passage of the pulse wave and the stroke volume of the ventricles. The condition of the vessel wall affects the pulse volume. Therefore in the present study we studied the changes in arterial pulse volume with age.

Methods: The study was conducted on 40 volunteers from general population having no apparent disease. The subjects were divided into two groups: Group- I and Group-II. The Pulse Volume was recorded by Pulse Transducer and Student Physiograph (Inco Ambala, India). Normal arterial pulse was recorded and the rate, rhythm and average amplitude for one minute were calculated. Ratio of averaged Pulse Amplitude (PA) and Mean Arterial Pressure (MAP) of each subject was also calculated.

Results: The mean age (years) of subjects in group-I was lower (19.30±1.25) as compared to group-II (61.60±5.91) ($p< 0.001$). The important finding of our study was the pulse amplitude (mm) (which represent the pulse volume) was significantly lower (7.24±1.36) in group II as compared to group I (19.10±6.87). We also calculated the ratio of Pulse amplitude and Mean Arterial Pressure which was significantly lower in group II as compared to group I.

Conclusion: Our data suggests that the pulse transducer and physiograph can easily record the pulse volume index and asses the cardiovascular risk.

Keywords: Pulse volume; Age; Arterial wall thickness; Physiograph

1. Introduction

Arterial wall thickness increases with age and is a strong predictor of cardiovascular risk in humans[1]. Aging is associated with a number of changes in cardiovascular structure and function. One such change that has potentially important physiological and pathophysiological implications is an increase in wall thickness of medium and large-sized arteries[2,4]. This increase in wall thickness is due to smooth muscle hypertrophy, which results in a thickening of the intima medial layer[3]. The measurement of arterial intimal-medial thickness (IMT) allow for the assessment of atherosclerosis in free-living population-based settings[5]. Measures of wall thickness are currently used as surrogate marker for and intermediate end points of atherosclerosis in observational studies and are also used to document regression or progression of atherosclerosis in clinical trials[6,7,8]. An association between increased carotid artery IMT and a more adverse cardiovascular disease (CVD) risk factor profile has also been documented[9].

Because atherosclerosis progresses over decades, epidemiological studies and intervention trials with clinical end points require long-term follow-up, participation of large populations, or both[10-13]. These requirements have to be met to provide data from which valid conclusions about the determinants of disease or the efficacy of a therapeutic intervention can be drawn[14]. As a consequence, such studies consume precious time and financial resources. To overcome these challenges, surrogate markers became the focus of intense attention[15]. Such markers might be used to investigate determinants of atherosclerosis at an early stage of the process and can, subsequently, assess modifiers of atherosclerotic disease progression, such as lifestyle and pharmacological interventions.

Moreover, the strength of a surrogate marker is enhanced by the fact that it may yield pathophysiological information at an early stage of the disease process. Surrogate markers, therefore, have an inherent value of their own. Some follow-up studies have confirmed a higher BMI during childhood to be associated with increased risk of coronary artery disease in adulthood[16-18].

Pulse volume refers to the movement of the vessel wall during the passage of the pulse wave. The degree of movement may be referred to as full, weak or thready. The stroke volume of the ventricles and condition of the vessel wall affect the pulse volume.

Further the concept of impedance incorporates the relationship among pulse pressure, arterial elasticity and diameter. The peak load on the heart at a given cardiac output is determined by the impedance of the vessel wall.

Functional stiffness (Ep) is equal to structural stiffness (Y) multiplied by thickness of the vessel wall (h) and divided by its mid wall radius (r),

$$Ep = Y \times h/r$$

According to the Mackenzie *et al* distensibility of arterial wall is inversely proportional to the functional stiffness[31].

There has been much recent interest in the relationship between arterial stiffness and cardiovascular disease. Pulse pressure and pulse wave velocity, surrogate measures of arterial stiffness, indicate that arterial stiffness increases both with age and in certain disease states that are themselves associated with increased cardiovascular risk, including hypertension, diabetes mellitus, and hypercholesterolemia and end-stage renal failure[19]. As changes can be detected before the appearance of clinically apparent vascular disease, arterial stiffness may act either as a marker for the development of future atherosclerotic disease, or may be more directly involved in the process of atherosclerosis. Arterial stiffness may be measured using a variety of different techniques, although at present the majority of measurements are made for experimental and physiological studies rather than in clinical practice. However, it is likely that over the next few years measurement of arterial stiffness will become an increasingly important part of the process of risk assessment, and may possibly also improve the monitoring of therapy in patients with conditions such as isolated systolic hypertension. Now it becomes necessary for physicians both in primary care and hospital practice to understand the importance of arterial stiffness. Therefore in present study we evaluated the pulse volume in relation with age in apparently healthy subjects.

2. Material and Methods

2.1 Subjects

The study was conducted on 40 volunteers from general population having no apparent disease. The subjects were divided into two groups: Group- I (n=20) and Group-II(n=20). The inclusion criteria for group- I were age $\leq$ 20 years, BMI< 24 kg/m^2, having no history of any systemic disease, smoking, tobacco chewing, alcohol consumption and medication. The inclusion criteria for group II were same as group- I except age which was $\geq$ 50 years. The nature of study was explained and informed consent was obtained from each subject prior to participation in the study. A thorough history was taken and clinical examination was performed to rule out any obvious systemic disease. The height and weight of the subjects were recorded by standard methods. The subjects were advised to do not smoke or participate in strenuous activities for 24 hours before the test and should take any medications and eat and drink as they normally would.

2.2 Recording of Pulse Volume and Blood Pressure

The subjects were lying in supine posture during the test and 30 minutes rest was given to each subject. The Pulse Volume was recorded by Pulse Transducer and Student Physiograph (Inco Ambala, India). Student Physiograph is used for the recording of Bio-Electrical Potential e.g. EEG, ECG, ENG, EMG, Pulse, Respiration, Blood Pressure etc. After switching off the main, plug in the coupler, for recording pulse in the coupler housing. The pulse transducer was connected

to the subject by wrapping it around the index finger tip of left hand and the other end of the transducer was connected to the coupler. The physiograph stimulator (one pulse/sec) was connected to the physiograph and adjusted to record the time base. The console mains were put on desired paper speed (5mm/sec) and sensitivity (10mV). Normal arterial pulse was recorded and the rate, rhythm and average amplitude for one minute were calculated. Ratio of averaged Pulse Amplitude (PA) and Mean Arterial Pressure (MAP) of each subject was also calculated. The method of determining human blood pressure with Sphygmomanometer was based upon the procedure recommended by the Committee of the American Heart Association. The subjects were placed in a supine position. The subjects were placed at ease and time was given for recovery from any unusual recent exercise or apprehension. Blood pressure determination was not done after recent meals. The arm was bared and care was taken to avoid any constriction of the arm by clothing or interfering articles. The deflated bag and cuff was applied evenly and snugly but not too tightly around the arm with the lower cuff edge about one inch above the anti-cubital space and with the rubber bag over the inner aspect of the arm directly over the brachial artery. Then blood pressure was measured by auscultatory method.

2.3 Calculation of Pulse Pressure (PP) and Mean Arterial Pressure (MAP):

Pulse Pressure was calculated by the following formula:

Pulse Pressure (mmHg) = Systolic blood pressure – Diastolic blood pressure

Mean Arterial Pressure was calculated by the following formula:

Mean Arterial Pressure (mmHg) = Diastolic blood pressure + 1/3 of Pulse Pressure

3. Observation and Results

The aim of present study was to validate the relation of pulse volume with age of different groups by novel cost effective technique using pulse transducer and students Physiograph. Therefore in present study, the subjects were divided in two groups on the basis of their age. The mean age (years) of subjects in group-I was lower (19.30±1.25) as compared to group-II (61.60±5.91), this difference was statistically significant ($p< 0.001$). (Table-I) The mean weight (kg) of group –I and group-II was 60.00 ±7.38 and 65.30± 4.03 respectively which was not significantly different. No significant difference was observed in BMI, SBP, DBP and Heart rate ($p> 0.05$). The pulse pressure, which was calculated by subtracting the DBP from SBP, was also not significantly different. The important finding of our study was that the pulse amplitude (mm), which represent the pulse volume, was lower (7.24±1.36) in group II as compared to group I (19.10±6.87) and statistically significant ($p<0.0001$)(table I,fig.I). The pulse amplitude in correlation to age, recorded by Student Physiograph is unique finding of our study. We also calculated the ratio of Pulse amplitude and Mean Arterial Pressure to nullify the effect of Stroke volume on the Pulse volume. The PA / MAP ratio was significantly lower in group II as compared to group I (Table I,fig.II).

Table – I. Study of different parameter in relation with Functional arterial stiffness

Parameters	Group-I(n=20)	Group-II(n=20)	p value
Age (years)	19.30±1.25	61.60±5.91	P<0.0001
Height(cms)	162.32±4.57	161.17±6.63	p>0.05
Weight (kgs)	60.00±7.83	63.30±4.03	p>0.05
BMI (kg/m2)	22.90±3.09	24.34±1.73	P>0.05
HR (bpm)	76.16±6.83	74.8±8.63	P>0.05
SBP (mmHg)	119.60±7.82	113.6±8.68	p>0.05
DBP (mmHg)	79.80±5.03	75.80±4.57	p>0.05
PP(mmHg)	39.8±6.7	39.8±7.21	p>0.05
MAP(mmHg)	93.03±5.22	87.04±5.12	p>0.05
PA(mm)	19.10±6.87	7.24±1.36	P<0.0001
PA/MAP	.205±.073	.084±.018	P<0.0001

BMI:Body Mass Index, DBP: Diastolic Blood Pressure, HR: Heart Rate, MAP: Mean Arterial Pressure, PP: Pulse Pressure, PA: Pulse Amplitude, SBP: Systolic Blood Pressure.

Figure-I comparison of pulse amplitude (PA) in both groups

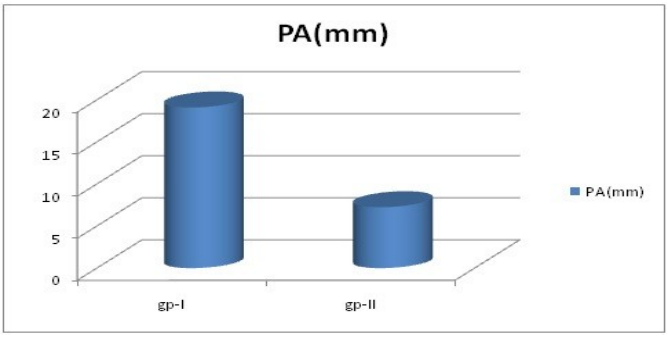
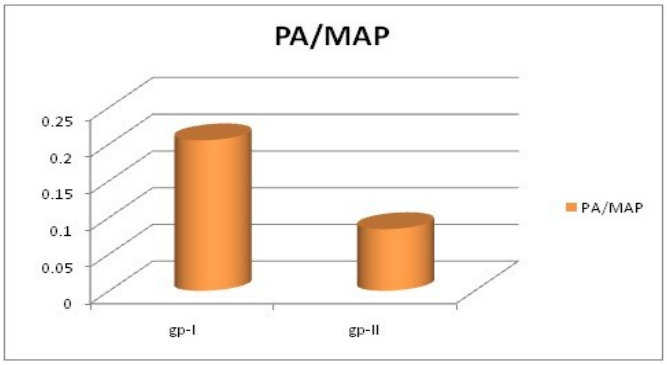


Figure-II comparison of ratio of PA and MAP



4. Discussion

The present study has been planned to assess the arterial wall stiffness by using a non-invasive technique (pulse transducer and student physiograph). A number of techniques are available for the assessment of elasticity of arterial tree. Some techniques give information on systemic arterial stiffness, while others only give information on local stiffness of the vessel being studied. The pulse pressure depends on the cardiac output, large-artery stiffness and wave reflection. The power of a surrogate marker is amplified by the fact that it may yield pathophysiological information at an early stage of the disease process[22]. Bramwell and Hill (1922) confirmed that the pulse pressure is a surrogate marker for arterial stiffness[20]. Elasticity can also be measured by pulse wave velocity by using magnetic resonance imaging (MRI) technique, but it is costly, time-consuming and can only be applied to large arteries[21]. Ultrasound can be used to measure arterial distensibility and compliance, but its use is limited to the larger and more accessible arteries. The technique also depends on the ability of the operator to image the walls of the vessel being studied precisely[22]. Angiography and Doppler ultrasound give the information about the condition of vessel wall, but they do not offer useful information on the early stages of arterial wall thickening[23]. Both techniques are underprivileged in light of the Glagov effect of initial arterial wall remodelling in the course of atherosclerosis progression[24,25]. Magnetic resonance imaging (MRI) techniques have been used to measure vascular wall stiffness and it demonstrates the inverse relationship between aortic distensibility and age[26]. Pressure pulse contour analysis is another method which has been used to estimate arterial stiffness non-invasively[27,28]. Digital volume pulse (DVP) recorded by Photoplethysmography and is used to demonstrate changes relating to drug effects and aging[29, 30].

Damping of the peripheral pulse, and temperature-dependant changes in the peripheral circulation are drawbacks associated with DVP. The technique has the advantages of being relatively simple and easily portable. If it were to be validated, it would have potential uses in the clinical setting. Because arterial tree stiffness is associated with the development of cardiovascular diseases thus by determining it the cardiovascular diseases can be predicted in its early stage.

Our data suggest that the pulse volume is significantly higher in young age subjects as compared to old age subjects. PA/MAP ratio is also significantly higher in young age subjects as compared to old age subjects. The elasticity of arteries of healthy young subjects is more than that of older healthy subjects[2,4]. If mean arterial pressure keeps constant then the elasticity of the vessel wall will be reflected by pulse volume (i.e. pulse amplitude). Present study indicates that relationship between pulse volume (pulse amplitude) and age of subjects is inversely proportional to each other. So we can say that the arterial tree stiffness increased with age. Findings of present study are in closed agreement with previous studies[28,29,31].

The present technique has the advantages of being relatively simple and cost effective. If it were to be validated, it would have potential uses in the clinical setting and in the experimental physiology work to evaluate the effect of various drugs and physiological conditions in human being and animals as well. The present technique may also be useful for screening purpose of cardiovascular system because elasticity is surrogate marker of various cardiovascular diseases.

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