

Assessment of Pulse Wave Velocity and Augmentation Index in different arteries in patients with severe coronary heart disease

Irina Hlimonenko, Kalju Meigas, Margus Viigimaa, Kristiina Temitski

Abstract— The aim of this study was to assess pulse wave velocity (PWV) and augmentation index in different arteries in patients with severe coronary heart disease (CHD). Signal measurements were obtained from 28 subjects. Severe coronary heart disease was confirmed by coronary angiography. Aortic PWV and Augmentation Index were measured using the TENSIOmed Arteriograph. Four other pulse wave velocities (upper limb PWV₁, upper limb PWV₂, upper limb PWV₃ and lower limb PWV) were obtained using PowerLab 4/20T device. It was found that aortic PWV was significantly increased in the CHD group compared with that in the control group ($P<0.01$). Augmentation index was significantly increased in the CHD group compared with the control group ($P<0.01$). This study shows the strong association of aortic stiffness and atherosclerosis. Pulse wave analysis can provide inexpensive and noninvasive means for studying changes in the elastic properties of the vascular system with the coronary heart disease.

I. INTRODUCTION

Pulse wave velocity (PWV) is widely used non-invasive method for evaluating atherosclerosis. Increased PWV is a marker of atherosclerosis and has been shown to reliably predict presence and extent of atherosclerotic vascular disease [1]. Aortic pulse wave velocity (PWV) predicts mortality from cardiovascular disease, ischaemic heart disease and stroke. Recent study has demonstrated that brachial-ankle pulse wave velocity were all significantly higher in coronary artery disease patients as compared to patients without coronary artery disease.[2].

The endothelium modulates arterial stiffness, which precedes overt atherosclerosis and is an independent predictor of cardiovascular events. Dysfunction of the endothelium may be considered as an early and potentially reversible step in the process of atherogenesis. Numerous methods have been developed to assess endothelial status and large artery stiffness, including PWV and augmentation index measurements.

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Increased brachial-ankle PWV is associated with impaired endothelial function in patients with coronary heart disease [3].

Aortic PWV is an independent predictor of coronary heart disease and stroke in apparently healthy subjects. Aortic stiffness can lead to low diastolic blood pressure, thereby possibly limiting coronary perfusion. In the recent large population-based study performed in elderly subjects aortic stiffness was strongly and independently associated with coronary atherosclerosis [4].

Arterial wave reflection is a major determinant of left ventricular function, coronary perfusion and cardiovascular risk. We investigated whether arterial wave reflection may detect atherosclerosis of peripheral arteries in patients with documented coronary heart disease (CHD). Augmentation index is a marker of extensive extracoronary atherosclerosis in patients with coronary heart disease [5].

However, very little is known about associations between PWV measured at different arteries and atherosclerosis in coronary, brachial and femoral arteries. Recent study has shown that carotid-femoral PWV correlated positively with coronary artery calcium score and increased with incremental coronary calcification category [6]. Authors of this study have concluded that carotid-femoral PWV is a better indicator of atherosclerosis than either carotid-radial or femoral-posterior tibial PWV, and should be used preferentially in studies of atherosclerosis and in stratifying risk in clinical settings.

The aim of the present study was to assess PWV and augmentation index in different arteries in patients with severe coronary heart disease.

II. MATERIALS AND METHODS

A. Population and examination

Signal measurements were obtained from 28 subjects (13 male, 15 female); their mean age was 59,5 (range 24-81). Group of CHD patients consisted of 18 subjects registered in the Department of Cardiology of the Tallinn North-Estonian Regional Hospital from January to March 2007. Severe coronary heart disease was confirmed by coronary angiography. Twelve patients out of 18 have been suffered from myocardial infarction.

The control group consisted of 10 subjects who were considered as persons without any cardiovascular disease or complication.

Aortic PWV and Augmentation Index were measured using the TENSIOmed Arteriograph. Signals from

peripheral sites were measured using PowerLab 4/20T device and four pulse wave velocities were manually calculated (upper limb PWV1, upper limb PWV2, upper limb PWV3 and lower limb PWV). Measurements using two different methods were performed one after another. All measurements were performed in a laboratory conditions in the supine positions for about 15 minutes.

B. Measurement of Pulse Wave Velocity and Augmentation Index using TENSIO Med Arteriograph

The Pulse Wave Velocity and Augmentation Index were measured using the TENSIO Med Arteriograph (TensioMED Ltd., Hungary). The measurement is based on the fact that during systole, the blood volume having been ejected into the aorta generates pulse wave (early systolic peak). This pulse wave runs down and reflects from the bifurcation of aorta, creating a second wave (late systolic peak). The difference between first and reflected systolic waves (in msec) is related to the stiffness of the aorta. The difference of the amplitudes of the first and second systolic waves (Augmentation Index) depends on the tone of the peripheral arteries (endothelial function). On the basis of those characteristics aortic PWV can be calculated. By analyzing the amplitude of the reflected and the first wave, the augmentation index can be calculated. In case of endothelial dysfunction reflected wave amplitude is higher than the first (early or direct) one, because of the summarization of the the first wave with the high amplitude second (reflected) wave. According to the TENSIO Med Arteriograph data: optimal Augmentation Index is $< -30\%$ and Aortic PWV < 7 m/s. The Arteriograph measures endothel/vascular dysfunction by measuring total Augmentation Index (further Augmentation Index) and the stiffness of the aortic wall by measuring the Pulse Wave Velocity of the aorta (further in the text "Aortic PWV"). Those indices were gathered for both groups: CHD and control group subjects.

C. Measurement of signals using PowerLab 4/20T

The PowerLab 4/20T is a four-channel recording instrument. It measures electrical signals through the inputs on its front panel. The purpose of the PowerLab system is to acquire, store, and analyze data. The raw input signal is in the form of an analog voltage whose amplitude varies continuously over time. This voltage is monitored by the hardware, which can modify it by amplification and filtering. After signal conditioning, the analog voltage is sampled at regular intervals. The signal is then converted from analog to digital form before transmission to the attached computer. The Chart computer software usually displays the data directly; it plots the sampled and digitized data points and reconstructs the original waveform by drawing lines between the points. Digital data can be stored on disk for later retrieval. For piezoelectric signal measurements piezoelectric sensors (MLT 1010 pulse transducer, AD Instruments) were used.

During the study signals were simultaneously acquired from hand and then from leg. First, signals from hand were

registered; three major sites were used to attach sensors: fingertip, wrist and elbow. Then sensors were attached to the leg to the following sites: toe and popliteal space (Fig.1).

D. Manual Calculation of Pulse Wave Velocity in peripheral arteries

The pulse wave generated by the left ventricular myocardium contraction and pulse wave is propagated throughout the arterial tree at a speed determined by the elastic and geometric properties of the arterial wall and the blood density. PWV is calculated from measurements of pulse wave transit time and the distance traveled by the pulse wave between two recording sites: $PWV = \text{Distance (meters)} / \text{Transit Time (seconds)}$.

The waveform analysis was performed to calculate pulse wave velocity in different arteries. The waveforms were analyzed offline using PowerLab 4/20T software. The computer program displayed the incoming waveforms on the screen of the computer. Transit time was determined from the time delay between the two corresponding foot waves: the proximal (A) and the distal (B) pulse waveforms. The foot of the wave is identified as the beginning of the initial upstroke. The distance traveled by the pulse wave between the two measurements sites was measured over the surface of the body with a measurement tape. PWV was calculated on the mean basis of 10 consecutive pulse waveforms to cover a complete respiratory cycle.

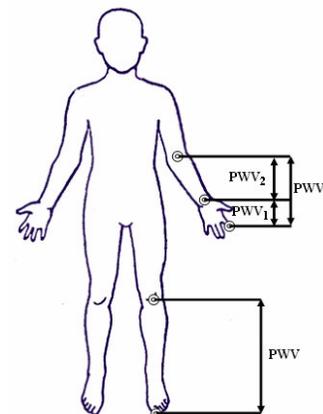


Fig. 1. Measurement sites of pulse wave velocity in different arteries.

The length of each the recorded signal was 15 minutes. The process of transition from one pulse to another was manual, which allowed editing of any poorly recognized pulse landmarks. The pulses were smoothed using a digital low-pass filter (50 Hz) and high-pass filter (8 Hz) to remove the low frequency baseline. Due to the patients' movements or irregular breathing anomalous pulses were rejected from the analysis. Based on the recorded signals 4 different pulse wave velocities were obtained: upper limb PWV1, upper limb PWV2, upper limb PWV3 and lower limb PWV. Further in the text for better understanding such indications will be used.

E. Statistical Analysis

All data analysis was performed using the program SPSS 10.0 for Windows. Data was expressed as the mean \pm SD. Multiple linear regression analysis (Pearson correlation) was used for the relationships between risk factors and PWV,

and between pulse wave velocity measured in different arteries. A value of $P < 0.05$ was considered statistically significant.

TABLE I
CLINICAL CHARACTERISTICS IN CHD AND CONTROL GROUP

	CHD (n=18)	Control group (n=10)	P value
SBP (mmHg)	150.0 \pm 26.8	122.0 \pm 14.8	<0.01*
DBP (mmHg)	90.5 \pm 13.6	70.5 \pm 6.9	<0.001*
Aortic PWV (m/s)	10.2 \pm 1.8	6.6 \pm 0.8	<0.001*
Augmentation index (%)	0.2 \pm 38.3	-73.3 \pm 20.4	<0.001*
Upper limb PWV ₁ (m/s)	6.8 \pm 2.0	5.2 \pm 1.9	NS
Upper limb PWV ₂ (m/s)	8.1 \pm 2.2	7.6 \pm 0.8	NS
Upper limb PWV ₃ (m/s)	7.8 \pm 1.4	6.4 \pm 1.1	NS
Lower limb PWV (m/s)	7.7 \pm 1.0	6.09 \pm 0.7	<0.01*

Data are mean \pm SD. SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure

* $P < 0.01$, difference is statistically significant.

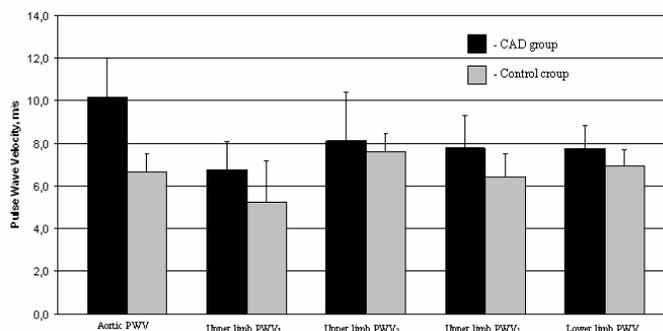


Fig. 2. Mean pulse wave velocity in different arteries in CHD and control group.

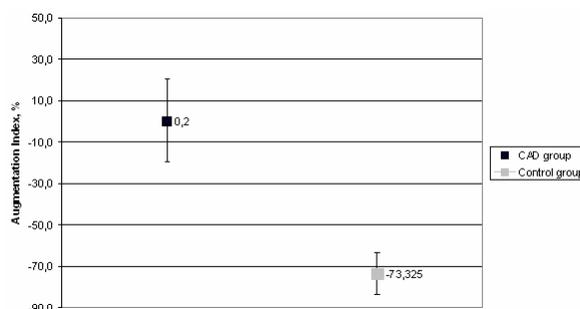


Fig. 3. Mean augmentation index in CHD and control group.

III Results

A. Comparison of CHD and control group

The clinical characteristics of our patients are summarized in Table 1. Patients with CHD were older, had a higher blood pressure and had more traditional risk factors. Aortic PWV was significantly increased in the CHD group compared with that in the control group.

What concerning peripheral pulse wave velocity only lower limb pulse wave velocity was significantly increased in the CHD group compared with that in the control group (7.7 \pm 1.0 m/s vs. 6.09 \pm 0.7m/s, $P < 0.01$) (Fig.2)

Also augmentation index was significantly increased in the CHD group compared with the control group ($P < 0.001$) (Fig. 3).

TABLE II
PEARSON CORRELATION COEFFICIENTS BETWEEN PULSE WAVE VELOCITIES IN DIFFERENT ARTERIES IN CHD PATIENTS (N=18)

	Aortic PWV (m/s)	
	Pearson correlation coefficient	p value
Upper limb PWV ₁ (m/s)	0,072	NS
Upper limb PWV ₂ (m/s)	0,501*	0,000
Upper limb PWV ₃ (m/s)	0,262*	0,000
Lower limb PWV (m/s)	0,231*	0,000

* P<0.01

TABLE III
PEARSON CORRELATION COEFFICIENTS BETWEEN PULSE WAVE VELOCITIES IN DIFFERENT ARTERIES IN CONTROL GROUP (N=10)

	Aortic PWV (m/s)	
	Pearson correlation coefficient	p value
Upper limb PWV ₁ (m/s)	0,123	NS
Upper limb PWV ₂ (m/s)	-0,168	NS
Upper limb PWV ₃ (m/s)	-0,134	NS
Lower limb PWV (m/s)	-0,293	NS

B. Correlation of Aortic PWV with peripheral pulse wave velocity in CHD patients group

In multiple regression analysis aortic PWV had a positive correlation with upper limb PWV₁, upper limb PWV₂ and lower limb PWV (P<0.01). The results are shown in Table 2.

C. Correlation of Aortic PWV with peripheral pulse wave velocity in control group

We also analyzed the correlation between Aortic PWV and peripheral pulse wave velocity in control group. The data are shown in Table 3. None of peripheral pulse wave velocities were significantly correlated with aortic PWV.

IV DISCUSSION

The objective of this study was to examine arterial stiffness in relation to atherosclerosis in different arteries in patients with severe coronary heart disease. Because endothelial dysfunction reflects a systemic vascular abnormality, we can indirectly evaluate the endothelial function of the coronary arteries by examining aortic stiffness or elastic properties of peripheral arteries.

The major findings of this study are that patients with coronary artery disease have increased aortic PWV compared with control subjects. That demonstrates that patients with CHD have impaired endothelial function compared with control subjects, and are consistent with previous studies [7, 8]. Control group subjects had

significantly less arterial stiffness (as reflected by both a lower PWV and a lower Augmentation Index).

The strong association of aortic stiffness with atherosclerosis at various sites of the arterial tree suggests that aortic stiffness can be used as an indicator of generalized atherosclerosis [9]. Not significant but still apparent difference between pulse wave velocity in peripheral arteries in patients with CHD compared with control group can indicate that changes of elastic properties of the walls of peripheral arteries less obvious or peripheral arteries undergo changes on the latest stages. To obtain significant difference group with greater number of patients should be investigated. One of the limitations of current study might be too small group.

However, difference of pulse wave velocity in lower limb was significant which can be explained by the fact that atherosclerosis is much more pronounced in lower limb arteries.

V CONCLUSION

As a result of our research we conclude that this type of analysis can provide a simple inexpensive and noninvasive means for studying changes in the elastic properties of the vascular system in patients with the coronary heart disease.

Further tests in a clinical environment are needed with more patients classified into groups to investigate correlation of pulse wave velocity with different stages of atherosclerosis.

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