Research Paper ■

Measurement and Analysis of Radial Artery Blood Velocity in Young Normotensive Subjects

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Abstract. In our study we analyze the velocity contour of blood flow in the radial artery and compare it with the typical parameters of the diastolic function of the left ventricle in young normotensive subjects with and without familial predisposition to hypertension. The analysis of velocity contour shows significant shortening of the time delay for two typical points on the secondary velocity wave: the maximum and the end of the wave. This shift of the secondary wave towards the primary wave shows diminished compliance of small vessels.

According to the results our conclusion is that changes in small vessels begin earlier than impairment of the diastolic function.

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Introduction

Abnormalities in the peripheral circulation make a mayor contribution to the circulatory disturbances seen in essential hypertension.[1] Reduction of arterial distensibility leads to: an increase in arterial impedance and, thus, in cardiac afterload; a reduction in diastolic pressure and in perfusion of organs such as heart; and an acceleration of arteriosclerosis due to an increase in systolic and pulse pressure and to a greater traumatic effect on the vessel walls. [2-12] Beside the mentioned, changes in the diastolic function of the left ventricle are among the earliest signs of developing hypertensive heart disease.[13] In our study we compare typical parameters which are drawn from velocity wave analysis of blood flow in the radial artery with parameters that determine the proprieties of the left ventricle during diastole in young normotensive persons with and without familial predisposition to hypertension.

Methods

The study comprises 32 young normotensive subjects, 13 of them with positive familial predisposition to hypertension. Echocardiographic examination includes measurement of early (E wave) and atrial (A wave) flow velocity through the mitral valve, computation of the deceleration time of the E wave (dt) and isovolumetric relaxation time. Also measured are the flow velocity propagation (Vp), the time delay of the peak E wave velocity from mitral tips to apex and the flow in pulmonary veins: systolic, diastolic, reverse atrial wave and the duration of this wave. Ejection fraction and left ventricular mass index are calculated from long axis view of the left ventricle. Flow velocity is measured using 10 MHz Doppler effect ultrasound probe (see Figure 1). To efficiently establish individual velocity pulses, R peaks are extracted from the ECG signal. The ECG R waves provide precise time markers for the velocity signal. The measuring system consists of an Echocardiograph (ECG), a Doppler effect ultrasound velocity meter (Doppler) and a portable computer. The used ECG device has an analog output signal which is sampled using a parallel port attached A/D converter with 12 bit data conversion resolution. The Doppler has an integrated 12 bit A/D converter and serial data communication capabilities, thus constantly sending sampled data with a fixed frequency of 100 Hz. Both mentioned devices are connected to a portable computer. Whenever valid data is received from the Doppler, the ECG data is read. This way the data from Doppler and ECG are captured synchronously. Such events occur with the period of 10 milliseconds.

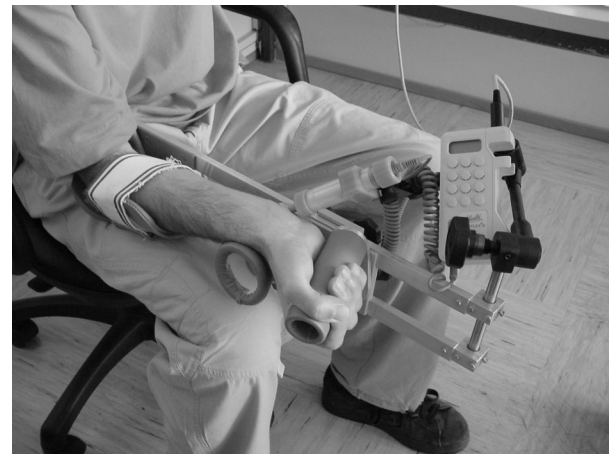

Figure 1 Custom designed arm attached Doppler velocity measurement probe holder

All analysis is carried out using the Matlab software package. The first step (pre-processing) of each measurement was to find and extract all R waves from the ECG signal. As presented in Figure 2, the R peaks are the splitting points of the synchronously captured velocity signal. Thus the result of every pre-processed measurement, is a large number of velocity wave sections (extracted between R peaks, see Figure 2). We call these sections 'velocity periods'. As the recording of each subject's data lasts five minutes, the number of extracted velocity periods varies from 280 to 400. The number of periods varies due to the heart rate frequency – with the R-R distance.

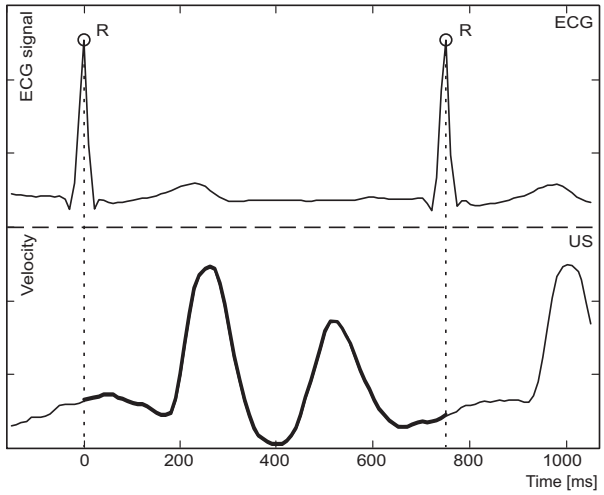

Figure 2 ECG signal and velocity measurement in radial artery

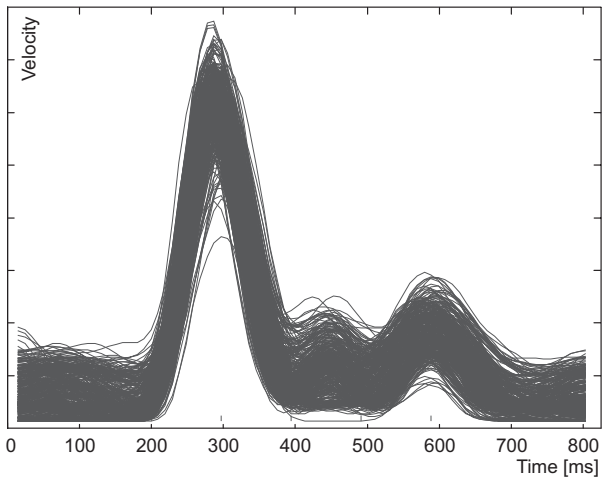

Figure 3 Extracted velocity periods aligned with starting points T0 (R peak); end points are shortened accordingly to the shortest R-R period

In Figure 3 all the extracted velocity periods from a single measurement are left aligned with their starting T_0 points (R peaks). As the R-R distance varies so does the length of individual velocity periods. To be able to display all the periods suitably they need to be right shortened, according to the shortest of the recorded periods. Because of the large dispersion of the velocity periods it is appropriate to calculate the mean period to read out the significant points easily (see Figure 4).

The mean velocity period is sufficiently described with six most significant points on the wave (T_0, …, T_5) and two inclinations of the primary wave (α_1, α_2) as presented in Figure 4.

The starting point on the mean velocity period is T_0 and matches exactly with R peaks. The point T_1 represents the start of the primary wave inclination. The maximum of the primary wave and also the global maximum of the mean velocity period is T_2. T_3 is the middle point between the primary and the secondary wave. The local maximum of the secondary wave is marked with T_4 and its ending point is marked with T_5.

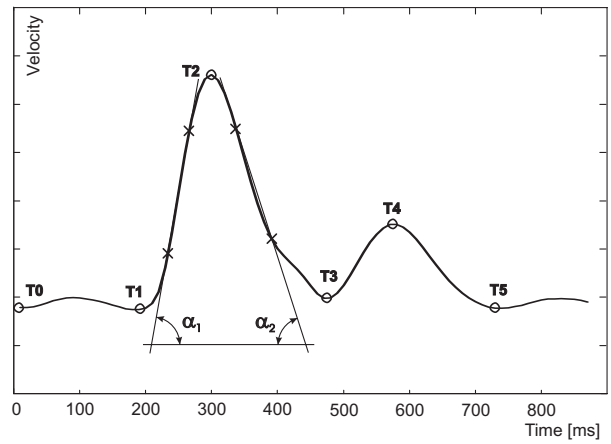

Figure 4 The characteristic points Ti and inclinations αj of the mean velocity period

The points T_1 and T_5, which mark the boundaries that separate the flat signal and the waves, are extracted by observing the slope change when the signal is close to its local minimum. Each characteristic point consists of two values, the value of the average velocity in that point and the time delay from the starting T_0 point. Further the inclinations of the rising and falling parts of the primary wave are calculated. The crosses in the Figure 4 represent the points that define the two inclinations. These points mark the 25% and 75% of the difference in amplitude between T_1 and T_2 for the rising and T_3 and T_2 for the falling edge of the primary wave. This way the inclinations of approximately linear segments of the primary wave are measured.

Results

The observed groups do not differ in parameters that describe diastolic function of the left ventricle i.e. E wave, A wave, dt of the E wave, Vp, IVRT and flow in pulmonary veins. However important differences in the time delay of points T_4 and T_5 regarding to starting point T_0 are found (see Table 1 and Figure 5).

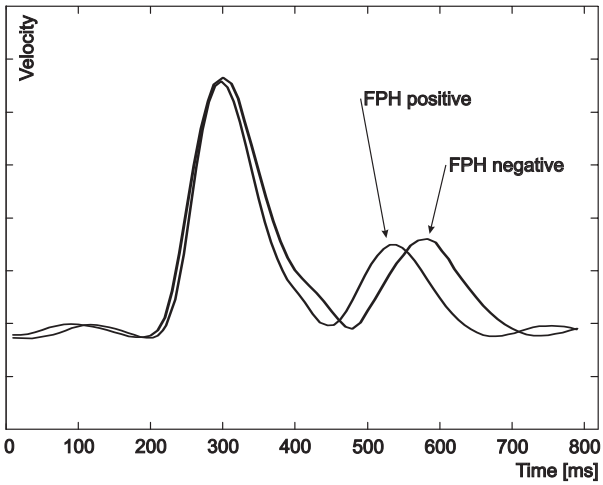

Figure 5 The shift of the secondary wave towards the primary in the FPH positive group

Table 1 Time delays for points T1, …, T5 according to T0 (ECG R peaks)

point	FPH negative	FPH positive	Sig.
T_1	180.8 ± 27.5	183.6 ± 15.7	NS
T_2	285.4 ± 29.3	280.0 ± 23.7	NS
T_3	457.7 ± 45.9	420.0 ± 46.5	0.059
T_4	573.1 ± 39.9	516.4 ± 68.2	0.019
T_5	744.6 ± 78.2	656.4 ± 105.2	0.028

FPH … familial predisposition to hypertension;
FPH column data … delay from the starting T_0 point, mean ± standard deviation [milliseconds];
Sig. … significance [t-test for equality of means].

Conclusions

Significantly shorter time delays for points T_4 and T_5 in the group with positive familial predisposition to hypertension are found. As the secondary velocity wave depends on the compliance of small vessels, the shift of the secondary wave towards the primary wave (i.e. smaller values T_4 and T_5) may be due to the structural or functional changes that are already present in small vessels of young normotensive subjects with familial predisposition to hypertension. The diastolic dysfunction of the left ventricle is composed of two main patophysiologic components: relaxation and compliance of the left ventricle.[14] Several authors found impaired relaxation and diminished compliance of the left ventricle in young hypertensive patients. Surprisingly enough our results do not show any difference in parameters that determine the diastolic function. We explain this by the fact that both groups are normotensive, so the left ventricle is not exposed to high pressure. On the other hand, the parameters, which are generally accepted and are used to assess the diastolic function, are not sensitive enough to distinguish both groups of normotensive subjects. On the base of our results we speculate that changes in small vessels occur before the alterations in relaxation and the compliance of the left ventricle in young normotensive subjects with familial predisposition to hypertension.

Medical aspect

Here we can stress again that there is a significant difference in the time delay of characteristic points on the secondary wave (T_4 and T_5) in the positive familial predisposition group. The shift of the secondary wave towards the primary wave is the consequence of diminished compliance of small arteries. There is no difference in parameters that determine the diastolic function. According to the presented results, we conclude that in young normotensive subjects with familial predisposition to hypertension changes of small vessels begin earlier than impairment of the diastolic function.

Technical aspect

During the research, we came across several ideas how to improve the system. The first step is to upgrade the system to be able to perform

significant point extraction and frequency analysis of velocity periods in real-time, during the data recording of each subject. In this way we intend to overcome the averaging of extracted velocity periods and perform the analysis on each extracted velocity period *on-the-fly*. Further we intend to add a new device that will enable us to perform blood pressure measurement synchronously with ECG and blood velocity measurement. This will provide our analysis with another data source that will improve the accuracy and broaden the field of our research.

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