AMPLIFICATION OF PERIPHERAL ARTERIAL PRESSURE IN YOUNG NORMOTENSIVE INDIVIDUALS

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ABSTRACT

The systolic and pulse pressure (PP) amplification in the peripheral arteries is explained with differences in the arterial wall structure and the reflection of pulse waves from points of branching and structural or diameter mismatch. Pressure amplification is much more manifested in young individuals than in middle aged or elderly persons.

Our aim was to clarify the mechanisms of pressure amplification in young normotensive individuals. Thirty-three individuals aged 19-23 years were examined using radial applanation tonometry. Central aortic pressure was evaluated using the Sphygmocor (AtCor Medical) software and pulse wave analysis. The amplification ratio (estimated as the ratio between peripheral and central PP) was 1.69 ±0.02. We have evidenced a negative correlation between the amplification ratio and the augmentation pressure in the aorta ($r = -0.703$, $p<0.0001$) as well as between the amplification ratio and the augmentation index (AI%75) ($r = -0.76$, $p<0.001$). This high negative correlation supports the idea that the larger amplification of peripheral systolic and PP in the young individuals results from the delayed return of the reflected pulse wave and its superposition on the forward wave during the early diastole. In the older persons with the development of structural alterations in the arterial wall pulse wave velocity increases and the reflected wave returns earlier, during systole. As a result the central PP, augmentation pressure and AI%75 become bigger while the amplification ratio decreases.

In conclusion, brachial arterial pressure is less informative in young individuals since it differs significantly from the central aortic systolic and pulse pressure.

***Key words**: pulse wave analysis, augmentation pressure, amplification ratio, applanation tonometry, cardiovascular risk*

Introduction. The recent introduction of a modern non-invasive technique – applanation tonometry – made possible our progress in understanding the intricate relationship between cardiac and vascular function. Cardiac load depends to a large extent on the vascular function: the afterload. During ejection left ventricular contraction should overcome the aortic systolic pressure. On the other hand aortic diastolic pressure is responsible for coronary perfusion and oxygen delivery to the myocardium [19]. Applanation tonometry made possible the computation of the aortic pulse wave based on data acquired from the radial artery using the validated generalized transfer function [6, 5]. The analysis of the central pulse wave (PWA) provided useful information concerning the arterial wall condition and cardiac load [1, 13, 9, 16].

The aortic pressure waveform (Fig. 1) is the result of the displacement of the compliant aortic walls when the stroke volume is ejected [10].

This displacement produces the first peak of the aortic pulse wave (P1), which results from the 'incident' or 'forward' wave. As it is well known the aortic wall deformation is propagated under the form of a high speed travelling pulse wave along the branching circulation. The pulse wave velocity (PWV) is much higher as compared to blood flow velocity and is calculated according the Moens-Korteweg equation as

$$PWV = \frac{\sqrt{\mathrm{E\,x\,h}}}{2r\rho},$$

where E is Young's elastic modulus, h is vascular wall thickness, r is vascular radius and ρ is blood density.

Therefore, the PWV gradually increases in distal direction, in the vessels, that are of a smaller diameter and with a relatively thicker wall. In addition, when the pulse wave reaches points of branching or points of mismatch – transition from elastic to muscular type of arteries, smaller

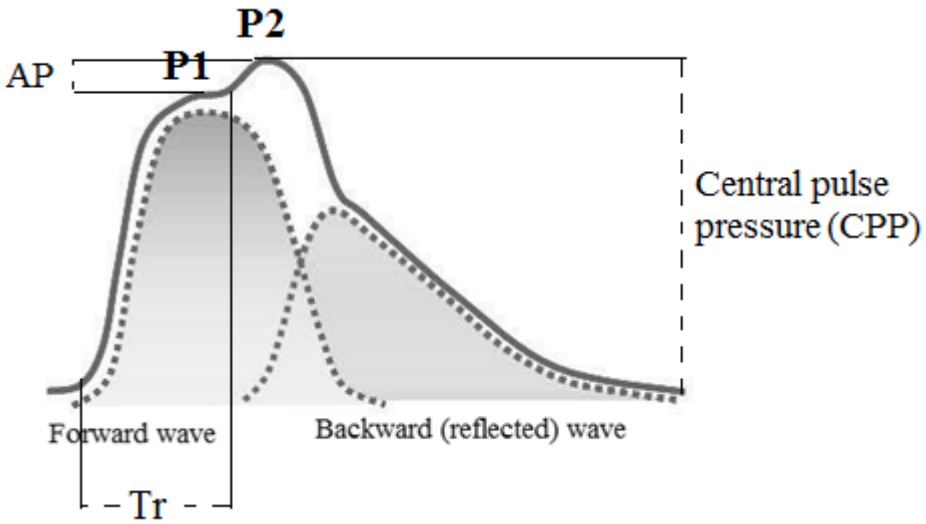

Fig.1. Aortic pressure waveform. The contribution of the forward and reflected waves to the generation of the systolic peaks P1 and P2 is shown. AP – Augmentation pressure. Tr - Time to return of the reflected wave.

diameter (higher resistance), etc., pulse wave is reflected and travels backwards towards the aortic valve. In fact, a large number of pulse waves are reflected and finally summated in one cumulative 'backward' wave, which returns back and is superimposed on the incoming 'forward' wave generating the second or P2 peak of the systolic part in the aortic waveform (Fig. 1) [1].

The amplitude of P1 in general depends on the stroke volume and on the elasticity of the aortic wall. The timing of the reflected wave return depends on a variety of factors. Some of them are pre-determined – e.g. the height of the individual; others are variable – e.g. the heart rate, which defines precisely when during the cardiac cycle the backward wave will return [20]; finally, the arterial wall properties determine PWV through E, the elastic modulus in the above formula. Arterial stiffening is a 'physiological' consequence to aging; its progress is accelerated under the effect of pathological processes like atherosclerosis. Arterial stiffness increases PWV and therefore modifies the timing of pulse wave return [9, 17, 7, 10, 3].

In summary, shorter individuals, slower heart rate and elevated PWV elicit early return of the backward wave. It returns during systole and generates a high P2. The difference between P2 and P1 is defined as the augmentation pressure (AP), which has a positive value in the older individuals (Fig. 1), while in the young healthy subjects AP is negative. Another important parameter of PWA is the augmentation index (AI%), which is calculated as $\frac{Augmentation\ pressure}{Pulse\ pressure}$ x 100 %. The augmentation index is often used normalized for heart rate 75 beats per minute (bpm). Another important parameter of PWA is Tr – the time to return of the reflected wave (Fig. 1) [2, 8, 17].

The 'physiological' and pathological alterations in central hemodynamics, higher systolic and lower diastolic pressure, lead to increased afterload during systole and diminished left ventricular coronary perfusion during diastole [14, 19]. Moreover, higher systolic pressure is transmitted to the target organs causing increased pulsatility of pressure in the delicate capillaries and pathologic changes in the coronary, cerebral and/or renal circulature. These pathophysiological alterations underlie the relevance of PWV and pressure waveform indices as markers of cardiovascular risk [4].

The systolic and pulse pressure values in the peripheral (radial) artery are always higher than corresponding values in the aorta [18, 12]. The phenomenon might be explained by the closer location of the reflection sites to the distal place of the radial pulse waveform recording. Therefore,

the rapid return of the backward wave causes a significant augmentation of the peripheral systolic pressure. In addition, the morphological characteristics of the more distal muscular and of a smaller caliber arteries predefines a higher PWV as compared to the aortic PWV that contributes to the early return of the reflected wave and to the augmentation of the peripheral systolic pressure as well (Fig. 2) [10]. On the other hand, diastolic pressure shows almost no difference between the central arteries and the radial site. The reason is that arterial vessels in between the heart and the radial artery are of a large diameter that does not exert significant resistance and does not cause a significant pressure fall. Hence, pulse pressure is also markedly amplified. This phenomenon is assessed using a parameter termed amplification ratio and calculated as $\frac{RPP}{CPP}$, where RPP is radial pulse pressure and CPP is central pulse pressure.

The aim of our study was to assess the parameters of the central systolic arterial pressure and to clarify the mechanisms of pressure amplification in young, healthy and normotensive individuals.

Materials and methods. Thirty-four young individuals aged between 20 and 23 years, females and males being equal in number, were studied. All examined subjects signed their informed consent to participate in the study.

The studied individuals rested for adaptation in supine position for 10 minutes. Brachial blood pressure was measured by means of a mechanical sphygmomanometer. Radial applanation tonometry was carried out using the Sphygmocor device (AtCor Medical, Sidney, Australia) (Fig. 2).

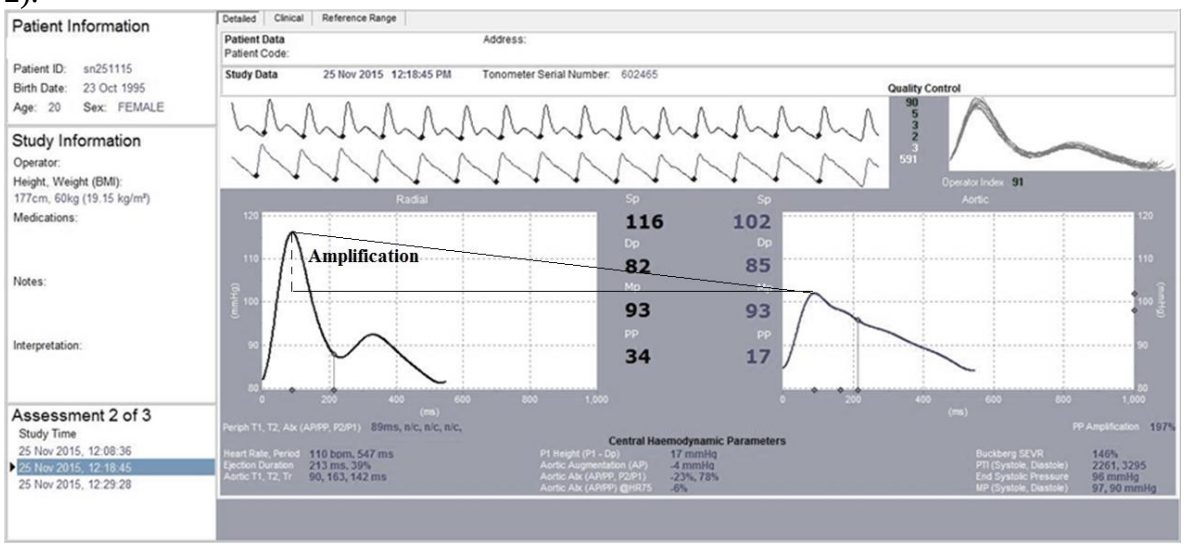

Fig. 2. A screen from the Sphygmocor device. The amplification of systolic pressure in the radial artery is clearly visible.

The basic parameters of the central and peripheral pulse waveform were assessed: systolic, diastolic, mean and pulse pressure, central augmentation pressure, augmentation index, augmentation index, normalized for heart rate 75 bpm, amplification ratio, amplification as an absolute value, and time to reflection Tr.

Statistical analysis was performed using the Data Analysis ToolPak of the Excell software. T-test and regression analysis were applied, the multiple R coefficient (Pearson's coefficient) was calculated and the level of significance for p was assumed to be at least 0.05.

Results. Our data are presented in Table 1.

Table 1. General characteristics of the studied individuals. Data are presented as means ±

SEM. ** p < 0.0001, * p < 0.0001 vs. central values.

Age	20.7±0.2 years
Central (aortic) systolic arterial pressure (CSAP)	98.6±1.3 mm Hg
Central (aortic) pulse pressure (CPP)	25.9±1 mm Hg
Central (aortic) augmentation pressure (CAP)	-0.38±0.5 mm Hg
Augmentation index, normalized for heart rate 75 bpm (AI%75)	-3.38±1.9 %
Radial systolic arterial pressure (RSAP)	115.3±1.8 mm Hg**
Radial pulse pressure (RPP)	43.6±1.8 mm Hg*
Amplification ratio	1.69±0.02
Amplification pressure	17.7±0.89
Time to reflection (Tr)	150±3.6 ms

The marked difference between the central and radial systolic and pulse pressures is demonstrated in Fig. 3.

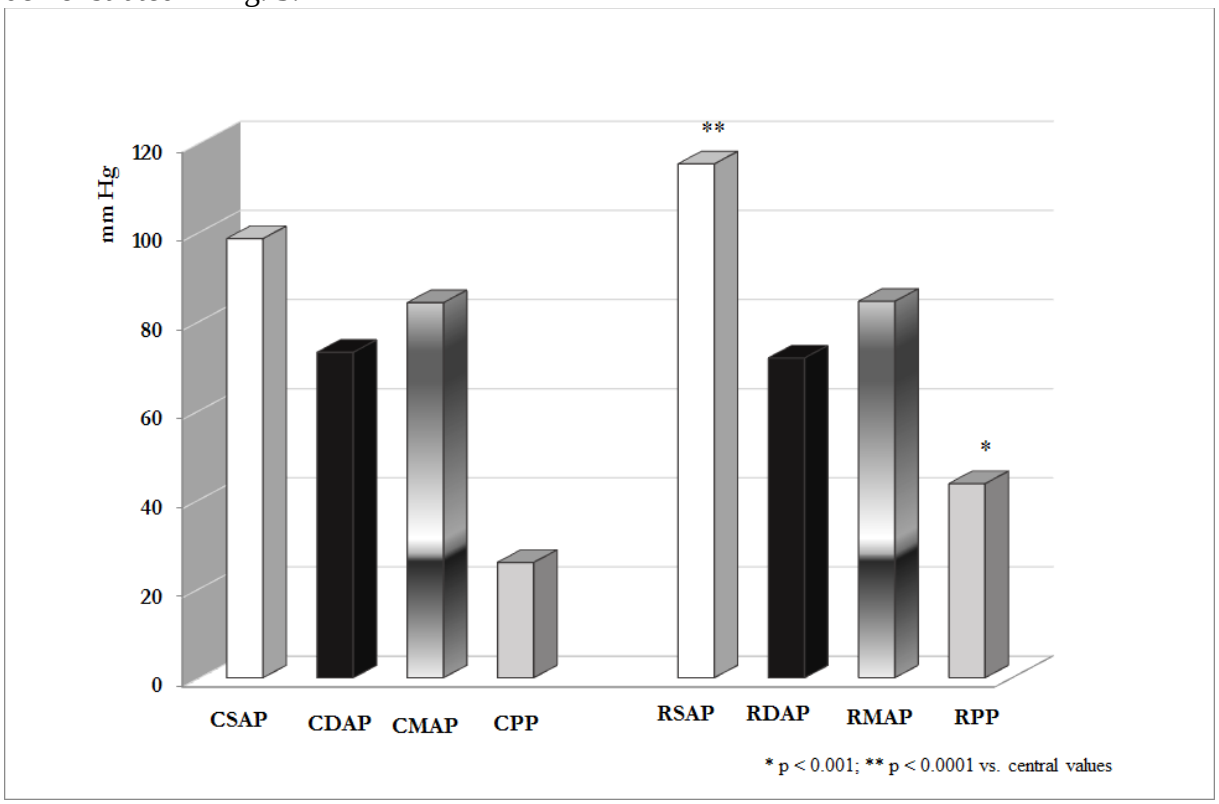

Fig. 3. Aortic and radial arterial pressures. CSAP – Central systolic arterial pressure; CDAP – Central diastolic arterial pressure; CMAP – Central mean arterial pressure; CPP – Central pulse pressure; RSAP – Radial systolic arterial pressure; RDAP – Radial diastolic arterial pressure; RMAP – Radial mean arterial pressure; RPP – Radial pulse pressure.

The correlation between central augmentation pressure and augmentation index, normalized for heart rate 75 bpm, on one hand, and the amplification ratio on the other is shown in Figures 4 and 5.

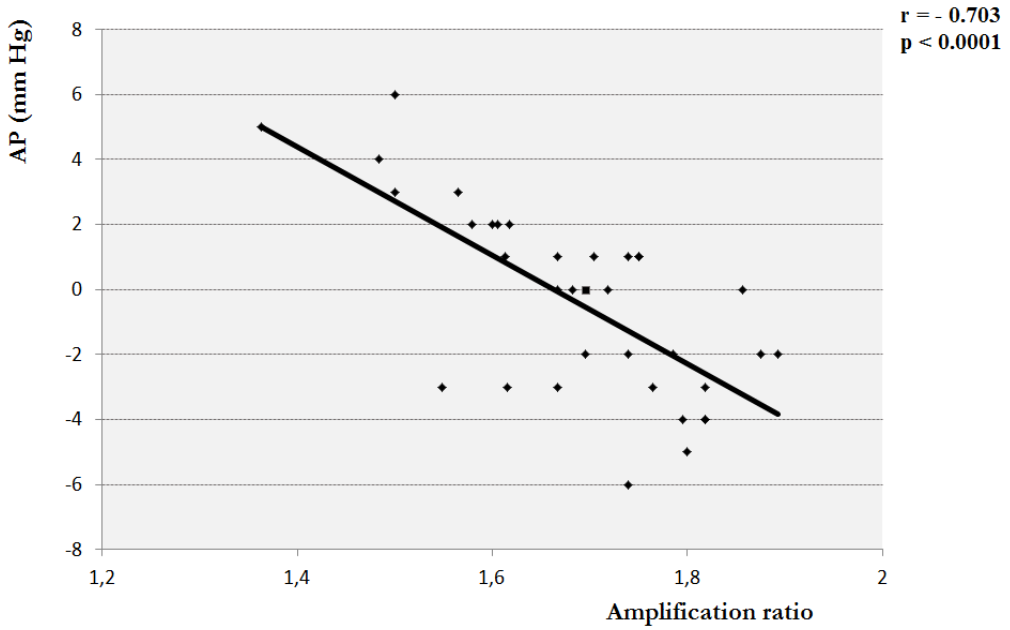

Fig. 4. Relationship between the amplification ratio and the augmentation pressure (AP). The linear regression line is shown (r = -0.703, p < 0.0001).

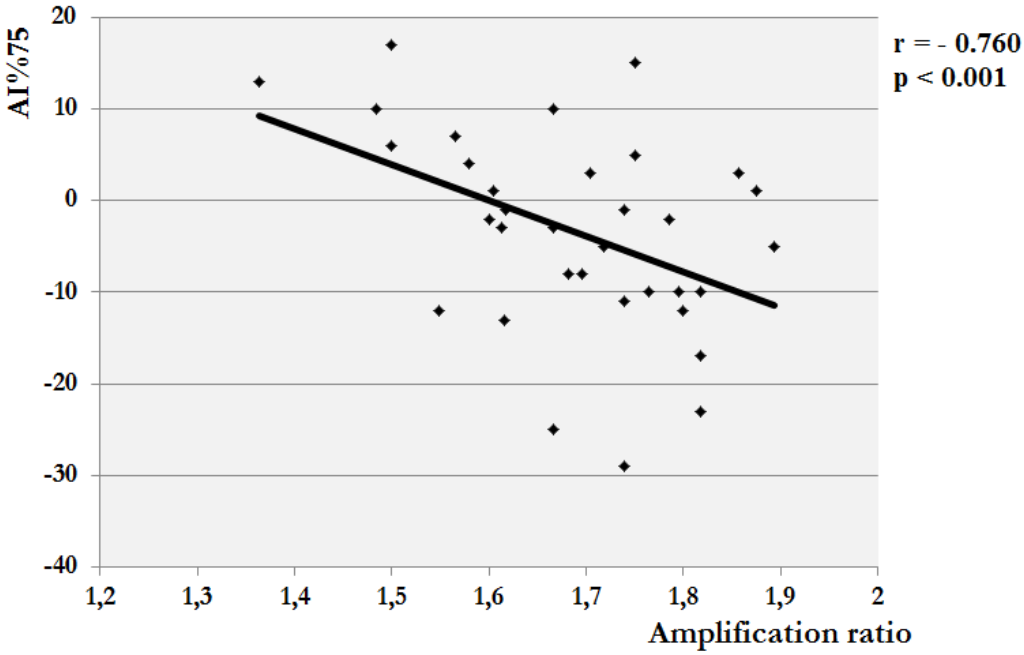

Fig. 5. Relationship between the amplification ratio and the augmentation index, normalized for heart rate 75 bpm (AI%75). The linear regression line is shown (r = -0.760, p < 0.001).

Discussion. Based on the PWA data we have demonstrated that the augmentation pressure and the augmentation index in our young adults were negative, i.e. the return of the backward wave appeared late in the cardiac cycle. This pressure waveform pattern is known as the C type of the central pulse waveform. Our data clearly showed that a large and statistically significant difference

existed between the central and peripheral systolic and pulse pressures ($p < 0.0001$ for systolic and $p < 0.001$ for pulse pressure) in the homogenous group of young and normotensive individuals. Expectedly, the amplification ratio (1.69±0.02) and the amplification as an absolute value (17.7±0.89 mm Hg) were large. These values completely corresponded to the data from the Anglo-Cardiff Collaborate Trail II [11], which was an epidemiological study carried out on a huge population (5648 individuals).

To clarify the underlying mechanisms of peripheral pressure amplification we have looked for the relationships between the amplification ratio and the augmentation pressure. We have found out the existence of a statistically significant negative correlation shown by the application of regression analysis. The correlation coefficient was high – $r = -0.703$, $p < 0.0001$. This finding confirmed the concept that the earlier in the course of the cardiac cycle returned the reflected wave (causing larger augmentation of the central systolic pressure) the smaller should be the difference between the radial and the aortic pressures. Similar relationship was demonstrated between the amplification ratio and the normalized for heart rate 75 bpm augmentation index – $r = -0.760$, $p < 0.001$. The latter correlation provided additional information; it showed that the negative relationship between the central augmentation and the amplification ratio existed even if normalized for heart rate 75 bpm. We may speculate that the inverse relationship between the amplified peripheral systolic value and the augmentation in the aorta mostly depended on the characteristics of the arterial wall, i.e. on the arterial stiffness and PWV. Therefore, we might assume that the amplification ratio is an additional marker for arterial stiffness and for cardiovascular risk. Our assumption is in conjunction with the findings of Bots and coll. [15] for a higher cardiovascular risk related to decreased pulse pressure amplification in older persons.

In conclusion, applanation tonometry is an informative and non-invasive method for evaluation of the cardiac and arterial functions. In addition to the known indices of PWA such as augmentation pressure, augmentation index, time to reflection, the amplification ratio is a promising parameter for cardiovascular risk assessment as an additional marker for the condition of the vascular wall in the large elastic conduit arteries.

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