Abdominal aortic aneurysm influences the indices of arterial stiffness recorded by pulse wave analysis

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Abstract

Background: Abdominal aortic aneurysm (AAA), forming a blood reservoir alters the geometry of the aorta, which along with increased stiffness of the aortic wall modifies central blood pressure wave, especially the reflected wave. The aim of the study was to compare indices of arterial stiffness recorded by pulse wave analysis (PWA) between patients with AAA and controls.

Material and methods: Sixty-nine patients (from 75 originally included) with asymptomatic AAA and 69 (from 74) age-, sex- and body mass index (BMI)-matching patients as a control group were analysed. The following variables of PWA recorded by applanation tonometry were evaluated: central pulse pressure (CPP), central systolic (CSBP) and diastolic (CDBP) blood pressure, central augmentation index (CAIx), the time from the beginning of a pulse wave to: the first systolic peak (CT1), to the beginning of the reflected wave (CT1R) and to the second systolic peak pressure (CT2).

Results: Patients with AAA had higher CAIx [33% (12) *vs.* 28% (20); $p < 0.001$] than in the control group, lower CPP [36 mm Hg (10) *vs.* 45 mm Hg (24); $p < 0.001$), higher CDBP [79 mm Hg (14) *vs.* 73 mm Hg (13); $p = 0.017$) and no significant difference in CSBP [115 mm Hg (15) *vs.* 119 mm Hg (23); NS). Shorter CT1 [102 ms (9) *vs.* 106 ms (12); $p = 0.004$) and CT1R [133 ms (11) *vs.* 138 ms (13.5); $p = 0.04$) and longer CT2 (232 ms (36) *vs.* 217 ms (35); $p = 0.007$) were observed in patients with AAA. Data are presented as median with interquartile range.

Conclusion: Differences of central blood pressure variables suggest increased arterial stiffness in patients with AAA. Despite lower values of pulse pressure, overall pressure load of the returning wave is higher, which affect the afterload of the heart.

Key words: arterial stiffness; abdominal aortic aneurysm; pulse wave analysis; central blood pressure

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Introduction

Abdominal aortic aneurysm (AAA) is routinely diagnosed when the transverse abdominal aortic diameter reaches above 30 mm [1, 2]. The incidence of AAA is estimated as 6–8% in men and about 0.6–1.6% in women and is increasing with age and in case of smoking or family history of AAA [3–5]. The most serious and life-threatening complication of AAA is a rupture, with the mortality rate up to 80–90% in acute setting [6].

The presence of AAA increases the risk of cardiovascular (CV) events and death [7–9]. The incidences of coronary artery disease, myocardial infarction and heart failure are high in the population with AAA, including small aneurysms, with the diameter between 30 and 54 mm [7]. The postulated reasons for increased number of CV events are advanced atherosclerosis, increased aortic stiffness (AS), with elastin fibres loss and aortic calcification. It may lead to increased cardiac afterload and left ventricular hypertrophy [10–13].

Exacerbated AS in the arteries results in changes in cardiovascular haemodynamics, mainly by premature peripheral reflection of the blood wave [14]. As a result, systolic blood pressure (SBP) and left ventricular afterload are increased, causing higher myocardial oxygen demand, while diastolic blood pressure (DBP) may be decreased, therefore lowering coronary perfusion pressure [15, 16]. Moreover, AS is one of the determinants of vascular aging and is considered as an independent predictor of major cardiovascular events, including stroke, myocardial infarction and left ventricular hypertrophy [17, 18]. AS has a predictive value of cardiovascular and all-cause mortality, coronary artery events, and fatal strokes in populations with cardiovascular risks [17, 19].

AS can be evaluated noninvasively by pulse wave analysis (PWA) using applanation tonometry. The arterial wave form, detected on a peripheral artery, is converted to the central blood pressure variables via algorithms embedded in the PWA device. The method is well validated with the invasive assessment [20, 21]. As previously shown, central blood pressure better corresponds to CV events than peripheral measurements [19, 22]. One of the main AS variables, augmentation index (AIx) is an independent predictor of future CV events in patients with hypertension, coronary artery disease and renal insufficiency [23, 24]. Another variable, pulse pressure (PP), is also considered as an AS marker and is one of the major determinants of CV risk in population [25, 26]. Central PP has a better prognostic value for CV complications and target organ damage than peripheral PP [27–29].

When compared to healthy volunteers, patients with AAA seem to have increased markers of AS [30–32], but the results differ between studies [33]. Therefore, this important topic is still not fully investigated, and detailed central pressure variables have not been extensively examined in patients with AAA. Therefore, the purpose of this study was to compare PWA indices of arterial stiffness between patients with AAA and controls with normal diameters of the abdominal aorta.

Material and methods

Participants

Seventy-five patients diagnosed with AAA without symptoms were invited to participate in the study. AAA was defined as the aortic diameter of equal or more than 30 mm. Seventy-four age-, sex- and BMI-matching volunteers as a control group were also included. All participants gave a written informed consent before enrolment. The research was approved by the Local Ethics Committee. Patients scheduled for an elective or urgent surgical intervention were not eligible for the study. The exclusion criteria included diseases potentially affecting the PWA results: thoracic aortic aneurysm, Marfan's syndrome or significant arrythmia, especially atrial fibrillation. The basic demographic data were collected. The BSA was calculated according to the formula of Bois and Bois [34]. The aortic size index (ASI) was obtained by dividing the maximum transverse diameter of the aorta by BSA as proposed by Davies et al. [35].

Measurements of the aortic diameters

The assessment of the aortic diameters was performed by the ultrasound evaluation (Aloka Prosound Alpha 6 with the Convex 3,5 MHz transducer, Hitachi Aloka Medical Systems, Hitachi, Ltd, Tokyo, Japan) by a medical doctor experienced in ultrasonography. The transverse axis 'D-MAX' was defined as the maximum diameter transverse to the longitudinal axis of the aorta. The mean aortic diameter 'D-MEAN' was calculated as an arithmetic average value of the two transverse diameters. The longitudinal diameter of the AAA 'L-MAX' was defined as the length in the long axis of the AAA segment of the abdominal aorta.

Central blood pressure parameters evaluation

Within two weeks after the ultrasound examination, the blood pressure measurements were performed. After at least 10 minutes of rest in supine position, the peripheral systolic and diastolic blood pressure were measured twice by oscillometric method on brachial artery and the average value was calculated. For central blood pressure, the variables were measured by PWA using applanation tonometry method. Blood pressure wave form was recorded on radial artery and was converted to central blood pressure variables with commercially available device (SphygmoCor EM3, with applanation tonometer SPT-304 and software SphygmoCor Cardiovascular Management Suite CvMS version 8.0, AtCor, Sydney, Australia).

During the examination the following blood pressure variables were obtained: peripheral systolic blood pressure (PSBP), peripheral diastolic blood pressure (PDBP), central systolic blood pressure (CSBP), central diastolic blood pressure (CDBP), the time from the beginning of a pulse wave to the first systolic peak (CT1), the time from the beginning of the pulse wave to the beginning of the reflected wave (CT1R), the time from the beginning of the pulse wave to the second systolic peak (CT2), reflected rising time (RRT) — obtained by subtracting CT1R from CT2, peripheral pulse pressure (PPP) — the difference between PSBP and PDBP, central pulse pressure (CPP) — the difference between CSBP and CDBP, central pressure at CT1 (CP1), central augmented pressure (CAP) — the difference between CSBP and CP1, central augmentation index (CAIx) — the pressure difference between CSBP and CP1 expressed as the percentage of CPP, ejection duration (ED). The central (CMAP) and peripheral (PMAP) mean blood pressure was assessed by the device automatically as a true mean value of the pressures. Due to a well-known influence of heart rate (HR) on CAIx and CAP, a correction to standard HR of 75 bpm was calculated (CAIxHR75 and CAPHR75). Heart rate (HR) was continuously measured during all examination. Main central blood pressure variables are presented on Figure 1.

Statistical analysis

The obtained data was analysed for the normality of its distribution with Shapiro-Wilk test, for

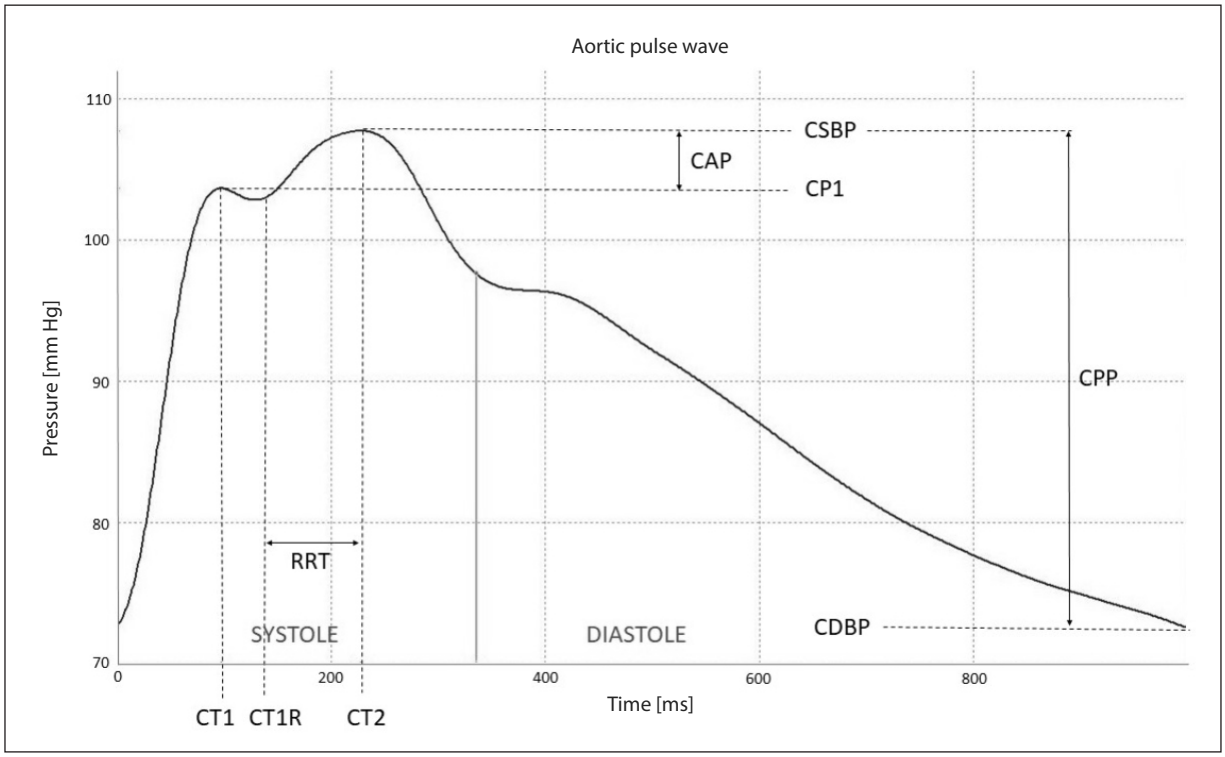

Figure 1. The original curve of the central pressure wave obtained by applanation tonometry. CSBP — central systolic blood pressure; CDBP — central diastolic blood pressure; CPP — central pulse pressure; CT1 — the time from the beginning of a pulse wave to the first systolic peak; CT1R — the time from the beginning of the pulse wave to the beginning of the reflected wave; CT2 — the time from the beginning of the pulse wave to the second systolic peak; RRT — reflected rising time; CAP — central augmented pressure; CP1 — central pressure at CT1

Table 1. Characteristics of the analysed abdominal aortic aneurysm (AAA) and control groups

Parameter	AAA group (n = 69)		Control group (n = 69)		p
	Median	IQR	Median	IQR	
Age [years]	72	12	73	12	0.985
Height [cm]	172	12	172	9	0.922
Weight [kg]	80	23	81	18	0.932
BMI [kg/m^2]	27.76	4.79	27.44	5.44	0.583
BSA [m^2]	1.96	0.29	1.97	0.21	0.992
HR [1/min]	63	13	63	16	0.853
D-MAX [mm]	43	9	20	3	< 0.001
D-MEAN [mm]	43	9.5	19	2	< 0.001
L-MAX [mm]	63	23	–	–	NA
ASI [mm/m^2]	22.42	4.57	10.12	1.25	<0.001

BMI — body mass index; BSA — body surface area; HR — heart rate; D-MAX — maximum diameter of the aorta; D-MEAN — mean value of the transverse diameters; L-MAX — AAA maximum length; ASI — aortic size index; IQR — interquartile range

each group (AAA and control) and each parameter separately. Half of the analysed data sets' distributions differed significantly from normal distribution ($p < 0.01$), (height, HR, D-MAX, D-MEAN, L-MAX, ASI, PSBP, PPP, CSBP, CPP, CAPHR75, CAIxHR75, CT1). Consequently, nonparametric statistical methods were used for all data for more adequate analysis. Differences of each variable between AAA and control groups were examined using Kruskal-Wallis tests. Statistical significance level of p-value was established as 0.05. For each variable medians and interquartile ranges were calculated. Analysis was conducted in R v. 3.10 (R Core Team, 2020).

Results

Seventy-five patients with AAA (including 57 men) and 74 patients of the control group (including 54 men) were primarily enrolled into the study. After initial verification of the quality of the measurements, results of 69 patients (including 53 men) for AAA group and 69 patients (including 53 men) for the control group were analysed. The basic characteristics of the analysed groups are presented in Table 1. The only significant differences were the morphologic parameters of the abdominal aorta and ASI.

The statistically significant differences in blood pressure variables between AAA group and the control group were found. PSBP, PPP and CPP were lower in the AAA group, while PDBP and CDBP were of higher values. In the AAA group, CAIx and CAIxHR75 were higher than in the control group. Comparing the times of the forward and the reflected waves, significant differences were also observed. CT1 and CT1R were significantly shorter in the AAA group while CT2 and RRT were longer in the AAA group. The values of the peripheral and central blood pressure variables are presented in Table 2.

Discussion

The main findings of our study are significant differences of central blood pressure variables: higher CAIx and CDBP, lower CPP, shorter CT1, CT1R and longer CT2 and RRT in patients with AAA.

The influence of the AAA on pressure can be a result of the geometric changes, within the abdominal aorta, but also of the increased arterial stiffness [36]. Our study supports the previously reported differences in CAIx and CAIxHR75 between patients with AAA and the control group without AAA [30, 32]. However, our study also shows, that patients with AAA have higher values of diastolic pressure — central and peripheral and lower peripheral and central pulse pressures when compared to the control group.

The reflected wave, which is directed to the heart, occurs in the ascending aorta after the main forward waveform. As a result, an additional pressure wave can be detected after systole [10, 37]. This can explain the increased DBP in patients with AAA. In vivo studies reported DBP as one of the factors significantly associated with AAA [38]. Additionally, DBP is considered as one of the factors involved in AAA development [38, 39]. The risk of AAA development is estimated to be 28% higher for every 10 mm Hg of DBP, even for values lower than typ-

Table 2. Central and peripheral blood pressure variables in both groups

Parameter	AAA group (n = 69)		Control group (n = 69)		p
	Median	IQR	Median	IQR	
PSBP [mm Hg]	120	15	129	27	0.002
PDBP [mm Hg]	78	14	73	13	0.019
PMAP [mm Hg]	92	15	92	18	0.853
PPP [mm Hg]	43	10	57	26	< 0.001
CSBP [mm Hg]	115	15	119	23	0.107
CDBP [mm Hg]	79	14	73	13	0.017
CMAP [mm Hg]	90	14.67	92	18	0.306
CPP [mm Hg]	36	10	45	24	< 0.001
CAP [mm Hg]	13	7	12	13	0.808
CAPHR75 [mm Hg]	10	4	9.5	7.25	0.685
CAIx (%)	33	12	28	20	< 0.001
CAIxHR75 (%)	30	9	22	14.25	< 0.001
CT1 [ms]	102	9	106	12	0.004
CT2 [ms]	232	36	217	35	0.007
CT1R [ms]	133	11	138	13.5	0.004
RRT [ms]	98	34	79	41.5	< 0.001
ED [ms]	325	42	314	36	0.293

AAA — abdominal aortic aneurysm; PSBP — peripheral systolic pressure; PDBP — peripheral diastolic pressure; PMAP — peripheral mean pressure; PPP — peripheral pulse pressure; CSBP — central systolic pressure; CDBP — central diastolic pressure; CMAP — central mean pressure; CPP — central pulse pressure; CAP — central augmented pressure; CAPHR75 — central augmented pressure corrected to heart rate 75 bpm; CAIx — central augmentation index; CAIxHR75 — central augmentation index corrected to heart rate 75 bpm; CT1 — central time from the beginning of the pressure wave to the first systolic peak; CT2 — central time from the beginning of the pressure wave to the second systolic peak; CT1R — central time from the beginning of the pressure wave to the beginning of the reflected wave; RRT — reflected rising time; ED — ejection duration; IQR — interquartile range

ically defined as hypertension (DBP < 90 mm Hg) [40]. As it has been previously established, DBP is at least as good predictor as SBP of vascular mortality in patients. Moreover, the associations of SBP and DBP with stroke and ischaemic heart disease are similar [41]. Both SBP and DBP are the risk factors for CV diseases and AAA, but DBP has even stronger connection with AAA than with CV diseases [38, 39].

In the mathematical model, AAA presence results in a reduce of PP. Swillens et al. investigated 5 different geometries of AAA and concluded, that the increasing diameter of AAA results in the reduction of PP [10]. This is an interesting observation, as far as exacerbated AS should lead to increased PP. It seems, that in case of AAA, the changes in the aortic geometry overcome the increase of arterial stiffness on the PP values. The shape of the aneurysm promotes perturbations of the blood flow as reported in biomechanical simulations and in vivo [10, 42, 43]. Our study confirms lower values of PP in patients with AAA.

Worth noticing is the fact, that the significant difference between peripheral systolic pressure was observed in our study, but the value of central systolic pressure did not differ. In healthy aorta, a physiologic amplification of the peripheral pressure is observed. The peripheral systolic blood pressure is up to 20–30 mm Hg higher than in the ascending aorta [44] (44). In case of aging and increased arterial stiffness this amplification decreases, causing similar values of the systolic pressure between periphery and the aorta. The unsignificant difference of CSBP along with significant difference of PSBP in our study, suggest exacerbation of stiffness in patients with AAA.

One can hypothesize, that all changes in central variables were caused by difference in PSBP; however, CSBP and CAP are dependent on peripheral measurements only in part. The other factors influencing the values are wave travel time and wave reflection coefficient [45].

The enhanced arterial stiffness in AAA is also indicated by changes of the times of the forward and reflected waves. CT1R is the time from the beginning of the pressure wave to the beginning of the reflected wave and evaluates the time after which the forward wave reflects in the periphery and returns to the ascending aorta. It is dependent mainly on the speed with which the pressure wave propagates and reflects. Despite in this study we did not measure pulse wave velocity (PWV), which is a marker of arterial stiffness, decreased CT1R indirectly confirms the higher PWV in patients with

AAA. As shown by some authors, PWV has greater values in the AAA group than in controls, but the results are incoherent with other studies [30–33, 46]. As far as the PWV is a marker of arterial stiffening, the decrease of CT1R could support the hypothesis of the decrease of the aortic wall compliance within the AAA segment.

On the other hand, time to the second peak of the pressure wave (CT2) is increased in the AAA group. The CT2 represents the time of the reflected wave's peak, which in stiffer arteries would probably be decreased, but the data are lacking. The explanation of significantly longer CT2 time in the AAA group is probably a widening of the abdominal aorta, which affects the reflected wave, causing an elongation of the reflected wave overall return time. The shortening CT1R and the elongation of the CT2 result in a decrease of the acceleration time of the pressure of the reflected wave (RRT), which increases the overall pressure load of the returning wave. RRT seems to be an important indicator of the pressure load of the returning wave [47]. Increased afterload of the heart has negative consequences on the systolic function. This is confirmed by the observation of Malm et al., that patients with AAA have significantly decreased systolic function assessed by global longitudinal strain and ejection fraction [48]. The time from the beginning of the pressure wave to the first systolic peak (CT1) defines the first phase of the systole, in which ventricles start to contract. The CT1 is dependent on the contractility of the left ventricle and the afterload. As has been shown by Malm et al. global longitudinal strain (GLS), which is a surrogate marker of systolic function of the left ventricle is slightly decreased in patients with AAA when compared to a control group [48]. Thus, the shortening of the CT1 cannot be affected by the increased contractility of the left ventricle but is a result of changes in the afterload. The elongation of CT2, suggests that the afterload is the most intense in the second phase of the systole. This causes a decrease of the pressure load in the first phase of the ventricle contraction. Decreased load enables the ventricles to initiate contraction easily and in short time. The shortening of CT1 could be classified as a marker of additional pressure load in the second phase of the systole but the data in the literature is lacking.

Limitations

The study has several limitations. The ultrasound evaluation allowed for the assessment of the very limited number of the dimensions of the AAA. Some studies suggest that other derivatives, such as velocity of the AAA, length of the lumen and intraluminal thrombus can play a significant role in the changes of the circulation hemodynamic and alter arterial stiffness parameters [49]. Therefore, a study using computed tomography angiography for measurements of the morphologic variables of the AAA could more precisely investigate these connections. Further analysis should be conducted to investigate the influence of the increased pressure load in patients with AAA on the function of left ventricle. A better understanding of the association of morphologic variables of AAA assessed by computed tomography angiography on haemodynamics of the circulation could lead to a better cardiovascular risk assessment in patients with AAA.

Conclusion

Abdominal aortic aneurysm significantly influences the shape of the central blood pressure wave. Differences of central blood pressure variables suggest increased arterial stiffness in patients with AAA. In contrast, changed geometry of the dilatated aorta results in significantly decreased pulse pressure. The increased overall return time of the reflected wave affects the pressure load on the left ventricle.

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