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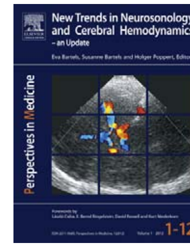

Arterial wall dynamics

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KEYWORDS
Arterial wall stiffness;
Distensibility;
Compliance;
Intima—media thickness;
B-mode;
M-mode ultrasound

Summary An early change in arterial wall dynamics introduced as a novel risk factor for cardiovascular events in various populations is discussed in this review.

Distensibility of an artery segment as reflection of the mechanical stress affecting the arterial wall during the cardiac cycle has been intensively studied recent years through the technological development of high-resolution ultrasound systems.

A decrease of arterial distensibility (i.e. increase of arterial wall stiffness) seems to be a common pathological mechanism for many factors associated with cerebrovascular and cardiovascular diseases. It is difficult to define the role of each factor affecting the arterial wall motions dependent mainly on the left ventricle, intra arterial pressure and blood volume, endothelium function, smooth muscle tone and neural control mechanism. The calculations of arterial compliance, elastic modulus, augmentation pressure, stiffness and intima—media thickness may help to identify the role of each mechanism if they are based on high-tech measurements of arterial wall.

The role of nervous regulation of blood vessel's tone in this process is not clear. Our studies show the strong correlation between autonomic imbalance and increase of carotid arterial distensibility in young patients. Various possible relationships between changes in the dynamic artery wall properties and neural regulation are discussed.

Methods of analysis of arterial wall motion

It is widely accepted that the early carotid arterial wall disease is a useful predictor of the risk of both ischemic stroke and coronary heart disease in asymptomatic population [1].

The parameters of arterial wall elasticity properties should be employed as a surrogate marker to detect early stage of vascular diseases. Increased artery wall stiffness and decreased arterial distensibility are accepted to be a common pathological mechanism for many factors associated with stroke, arterial hypertension, diabetes mellitus, hyperlipidemia and myocardial infarction [2,3].

Several quantitative or qualitative analysis methods for arterial wall function have been suggested. From them the most popular are the detection of flow-mediated dilatation (FMD) of brachial artery, assessment of peripheral arterial pressure waveforms, measurements of pulse wave velocity (PWV), measurements of arterial distensibility and stiffness with calculation of Young's modulus of elasticity of wall material, wall thickness and blood density.

Flow-mediated vasodilatation (FMD)

In the 1990s, high-frequency ultrasound imaging of the brachial artery to assess endothelium-dependent flow-mediated vasodilation was developed. Although FMD is widely used to provide the information about endothelium function in common it is related to the capacity to respond

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to different stimuli and confers the ability to self-regulate tone of the brachial artery only [4].

Pulse wave velocity (PWV)

Another assessment of arterial stiffness and compliance can also be performed by measurements of the speed of travel of the pressure pulse wave along the specified distance on the vascular bed. To measure PVW, pulse wave signals are recorded with pressure tonometers positioned over carotid and femoral arteries and are calculated as a ratio of distance and time delay:

$$\text{PWV} = \frac{\text{Distance}\,(D)}{\text{Time delay}\,(\Delta T)}\,\text{m/s}$$

Measurement of aortic PWV seems to be the best available non-invasive measurement of aortic stiffness while it is not specific for changes in elastic properties of carotid arteries [5—7,10].

Parameters of arterial wall distensibility and stiffness

Since no precise direct measurement method for the determination of arterial wall elasticity or stiffness has been suggested several indirect methods such as calculation of arterial compliance, Young's modulus of elasticity, stiffness index and arterial distensibility are commonly used.

The different parameters of carotid artery's wall elasticity could be measured by high resolution B-mode and M-mode ultrasound using manual and automatic measurements as well as wall echo-tracking system [8,9]. Development of methods based on ultrasound RF signal, tissue Doppler imaging and other tracking systems helps to increase the accuracy of automatic measurement of vascular wall properties such as IMT, arterial stiffness/distensibility and wall compliance, although even these methods are not free from errors [8,11,12].

The good reproducibility of carotid arteries diameters measured by 2D grayscale imaging, M-mode and A-mode (wall tracking) is proved [13]. However it is also mentioned that very small changes in linear measurements of carotid diameters can have big effects on estimates of arterial mechanical properties such as strain and Young's modulus. Additionally the cross-sectional imaging cannot be used to determine diameter or area of the lumen for a current clinical setting because of inadequate image definition of the lateral walls.

Carotid distensibility measured as changes in arterial diameter or circumferential area in systole and diastole is a reflection of the mechanical stress affecting the arterial wall during the cardiac cycle.

Distensibility can be calculated as $\text{Ds} - \text{Dd}$

where Ds is end-systolic diameter of artery. Dd is end-diastolic diameter.

$$\text{Distensibility or Wall Strain} = \frac{\text{Ds} - \text{Dd}}{\text{Dd}}$$

$$\text{Cross-sectional distensibility} = \frac{\text{As} - \text{Ad}}{\text{Ad}}$$

where As is the systolic cross-sectional area of artery. Ad is diastolic cross-sectional area.

It is difficult to understand and define the role of each factor influencing the arterial wall dynamics. Vasodilatation and vasoconstriction are dependent upon the left ventricle and intra arterial pressure and blood volume, endothelium function, smooth muscle tone and neural control mechanism.

Could the type of measurement and analysis of arterial wall distensibility help to define the mainly affected part of arterial wall involved in pathological process?

The influence of left ventricle function on a blood pressure could be measured by calculation of total arterial compliance:

$$\text{TAC} = \frac{\text{SV}}{\text{PP}}$$

where SV is left ventricle stroke volume.

Classical compliance is a change in blood volume in response to a given change in expanding pressure:

$$\text{CC} = \frac{\Delta V}{\Delta P} - \text{volume change to pressure ratio}$$

Since the distensibility of arterial wall is mainly blood pressure and volume dependent the systolic and diastolic pressure ratio is included in a most of calculations of vessel's elastic properties [14,15].

Wall stress can be defined as the difference in systolic and diastolic blood pressure:

$$\text{Pulse pressure (PP)} = \text{Ps} - \text{Pd}$$

The stress/strain relationship can be measured as vessel's diameter (or area) and pressure compliance given by different equations [16,17]. The most frequently used are:

$$\text{Compliance}\,(C) \quad C = \frac{\text{Strain}}{\text{PP}}$$

Pressure/strain elastic modulus (EM) is calculated as

$$\text{EM} = K \times \frac{\text{Ps} - \text{Pd}}{\text{Strain}}$$

where K is conversion factor for mmHg to Nm = 133.3.

Young modulus of elasticity (Y) which reflects the stiffness of an isotropic elastic material and can be defined as a ratio of stress to strain per unit area [18].

$$Y = \frac{\Delta P}{\Delta D} \cdot \frac{\text{Dd}}{\text{IMT}}$$

where IMT is intima—media thickness.

Stiffness index (β) is calculated as

$$\beta = \ln \frac{\text{Ps}}{\text{Pd}} \cdot \text{Strain}$$

Young elastic modulus (EINC)

$$\text{EINC} = \frac{3(1 = \text{LCSA/WCSA})}{\text{DIST}}$$

where LSCA — luminal cross-sectional area; WSCA — mean wall cross-sectional area; DIST — cross-sectional distensibility.

There are some beliefs that inclusion of different measurements of wall properties as well as hemodynamic parameters in equation could provide more informative and comprehensive index.

Like EINC-pressure and EINC-stress curves calculated from IMT and from diameter and pressure waveforms could provide more precisely direct information about elastic properties of the wall material that is independent of the vessel's geometry, whereas distensibility gives information on the elastic properties of the artery as a hollow structure [19].

The same could be said about the measure of contribution that the wall reflection makes to systolic arterial pressure. These measurements of reflecting waves coming from periphery to centre are calculated as augmentation pressure (AG) and augmentation index (AI) [20,21].

The disadvantage of above mentioned calculations lies in the comparison of elastic properties of different arteries like the comparison of wall dynamics of carotid artery to changes in blood pressure measured in a brachial artery. Calculations of FMD, PWV, Ai and other stiffness parameters cannot be attributed to carotid artery properties only since brachial, femoral, aortic and internal carotid arterial segments differ in the proportion of elastin—collagen to smooth muscle as well as proportion of endothelium to media layer and neural control.

Thus, the recording of pressure ratio during the cardiac cycle in a brachial artery can provide only indirect information of pressure/strain ratio in carotid artery. Considering this argument, it seems logical to evaluate carotid artery wall dynamics by ultrasound measurements of arterial wall structure and movements in a strictly precised vascular area.

Endothelium, smooth muscle and arterial distensibility

Apart from the blood pressure as the major determinant of vessels stretch the blood flow shear stress could play the important role in arterial distensibility. Endothelial cells are the primary vascular cells exposed to shear stress from the friction of laminar blood flow against the vessels wall. One of possibilities to detect the influence of endothelium and smooth muscle on arterial distensibility or stiffness is the recording of intima—media thickness (IMT) and its relation to vessels diameter (Fig. 1). The most popular are the measurements:

IMT to vessel's radius ratio:

$$\frac{\text{IMT}}{\text{Radius}} = 2 \times \frac{\text{IMT}}{\text{mean internal diameter}},$$

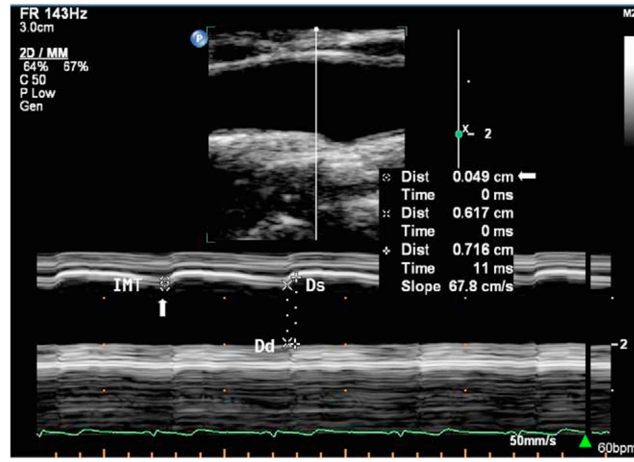

Figure 1 M-mode image of the right bulb of common carotid artery of 24 years old female with normal arterial blood pressure. IMT is 0.049 cm. The artery distension during cardial cycle: Ds — Dd = 0.716 — 0.617 = 0.1 cm, where Ds is marked with + Dd marked with ×.

Mean circumferential wall stress (MCWS) [19]

$$\text{MCWS} = \frac{\text{Mean BP} \times \text{mean internal diameter}}{2 \times \text{IMT}}$$

IMT and MCWS are indicative for changes in both endothelium and smooth muscle wall's layers since even high resolution ultrasound technique can provide the image of intima—media complex [22]. This technique with the phased tracking can obtain the measurements even of minute changes in IMT. From the maximum change in thickness during one heartbeat, the radial strain of each assigned layer in the artery wall (ε_r) is calculated as:

$$\varepsilon_r = \frac{h_{\max} - h_{\min}}{h_{\max}}$$

where $h_{\max}$ and $h_{\min}$ — maximum and minimum thickness of an assigned layer in the wall, respectively [23].

Commonly used the IMT measurement become the marker of early stage of decreased elasticity or increased stiffness of arterial wall. Significant correlations between increased IMT and the presence of arterial hypertension, hyperlipidemia, arterial atherosclerosis, diabetes mellitus and aging had been proven in many studies [24]. IMT and carotid artery stiffness became useful predictors of the risk of cerebrovascular and cardiovascular events [25].

Some results of the correlations between IMT and arterial distensibility indicate that gender- and age-related differences can be manifested even in young, healthy adults and may be identified with techniques that assess carotid distensibility across a range of pressures [26].

Although smooth muscle tone is a key determinant of mechanical properties of arteries its assessment in humans is technically limited and direct contribution of vascular smooth muscle to artery elastic mechanics is controversial [26,27]. Detecting the influence of tone on arterial properties is possible by applying sympathetic/parasympathetic stimulating test to the measurements of wall elastic properties.

Neural stimuli and arterial distensibility

There is the certain association between the changes in carotid arterial distensibility and autonomic imbalance. Some results of investigations suggest that the pathophysiological state of arterial distensibility may modify the autonomic balance. Carotid arterial distensibility is an important determinant of improvement in autonomic nervous regulation after the function of left ventricular wall motion abnormality has been improved [28]. All together both factors — changes in carotid distensibility and changes in left ventricular diastolic filling can influence carotid baroreceptors. Although it is known that baroreceptor sensitivity is reduced with increasing age and in patients with arterial hypertension it is difficult to determine whether this reduction is caused by reduction of arterial distensibility or disturbances in the neural transduction part of baroreflex arc [8]. Some data support the hypothesis that reduction in carotid artery wall elastic properties may lead to low vagal tone. Increased cardiovascular risk associated with low vagal tone may partly be mediated via changes in carotid artery elastic properties [29].

The hypothesis that carotid arteries undergo rapid changes in distensibility on moving from the supine to head-up tilt postures and, subsequently, that this change in carotid distensibility might be associated with concurrent reductions in cardiovagal baroreflex sensitivity had been tested [30]. It might be speculated that the reduction in diameter and maximal distensibility of the carotid region in orthostatic tests alters the interactive effects of the various types of baroreceptor afferents from the carotid sinus that differentially affect blood pressure control. Some findings indicate that sympathetic activation is able to decrease radial arterial compliance in healthy subjects. The reduction in arterial compliance probably resulted from complex interactions between changes in distending blood pressure and changes in radial arterial smooth muscle tone [31].

Values of rates of carotid distention are highly variable in young healthy individuals. There are also findings of carotid sinus distensibility exceeded aortic arch distensibility at the ages<35 whereas this relation was reversed at the ages >35. It could be assumed that this feature may impact on the ability to observe more consistent acute adaptations to postural perturbations [32]. These findings can also be explained by more pronounced effect of nervous regulation on arterial wall motion in young people. Furthermore the fact mentioned in the SMART study that some patients with the low systolic blood pressure had decreased arterial stiffness i.e. increased arterial distensibility coincided with our numerous observations in the practical survey of blood vessels and provoked the question whether it is a consequence of imbalance of autonomic regulation of wall dynamics [2,33].

Material and method

To detect the changes in the carotid artery wall tone we examined 97 young patients (42 men, 55 women from 17 to 35 years of age,) selected from patients who visited our hospital between 2002 and 2005 for clinical examinations. The main complaints were weather dependent and stress related headache, dizziness, excessive sweating, orthostatic light headedness, postural hypotension and fainting in history. All of the clinical routine tests had been done to exclude any disease which could cause above mentioned symptoms. Blood pressure instability during orthostatic test had been detected in the most of cases ($n = 78$) The tendency to low brachial blood pressure (s/d $101/54 \pm 12/9$ mmHg) found in 66 cases and slightly raised brachial blood pressure (s/d $140/75 \pm 9/7$ mmHg) in 12 cases. All patients underwent neck and cerebral blood vessels examination as a part of clinical tests. Results of ultrasound examinations of carotid artery had been compared with the results of the same examination of control group from 25 sex and age matched healthy individuals.

As a part of routine ultrasound examinations blood vessels of neck were examined usual way by 4—7.5 MHz linear probe and cerebral vessels by 3—3.5 MHz sectoral probe using two ultrasound systems — ''Applio'', Toshiba Medical Systems and ''iE-33'', Philips. Measurements had been done by one experienced examiner and data from both ultrasound systems had been compared. The small group of 7 patients was observed using both machines.

Ultrasound images of carotid artery were acquired and IMT measurements were done using B-mode regime usual way. Blood flow was examined using Color and Power Doppler mode in a standard regime. To register arterial wall's moving during cardiac cycle the M-mode was applied additionally to B-mode and Color-mode images. With a high M-mode resolution it was possible to define all layers of arterial wall and to measure IMT. All measurements of vessel's IMT and wall movement obtained from B-mode images and M-mode images had been compared and subsequent mean values had been calculated to avoid inevitable errors (Figs. 1 and 2). The area for measurements was carotid bulb dilation. The wall movements were measured as end-systolic (Ds) and end-diastolic (Dd) diameters of carotid artery (Fig. 1).

Results

There was a good comparability of measurements obtained using both ultrasound systems.

IMT of carotid artery of normotensive and hypotensive patients with a signs of autonomic nervous dysfunction did not differ from IMT of healthy controls (mean far wall CCA IMT 0.46 ± 0.07 mm, max -0.53 ± 0.08 mm) while patients with mild hypertension had higher rates of far wall CCA IMT (mean 0.54 ± 0.07 mm, max 0.65 ± 0.09 mm).

The carotid artery distensibility was significantly higher in a patient group as compared with a group of healthy controls: 0.11 ± 0.04 cm and 0.07 ± 0.02 cm respectively. The same change in distensibility in patients with initial mild hypertension was not statistically significant.

The peak systolic blood velocity in carotid artery ($V_{max} \pm$ sd 125 ± 15 cm/s) was increased compared to healthy individuals (V_{max} 87 ± 13 cm/s) Systolic acceleration was accompanied by increase of pulsative index (1.96 ± 0.87) as a result of drop of diastolic velocity (Fig. 2).

Signs of impaired arterial wall tone occurred as vascular bruits and heart tone registered not only in dopplerograms of

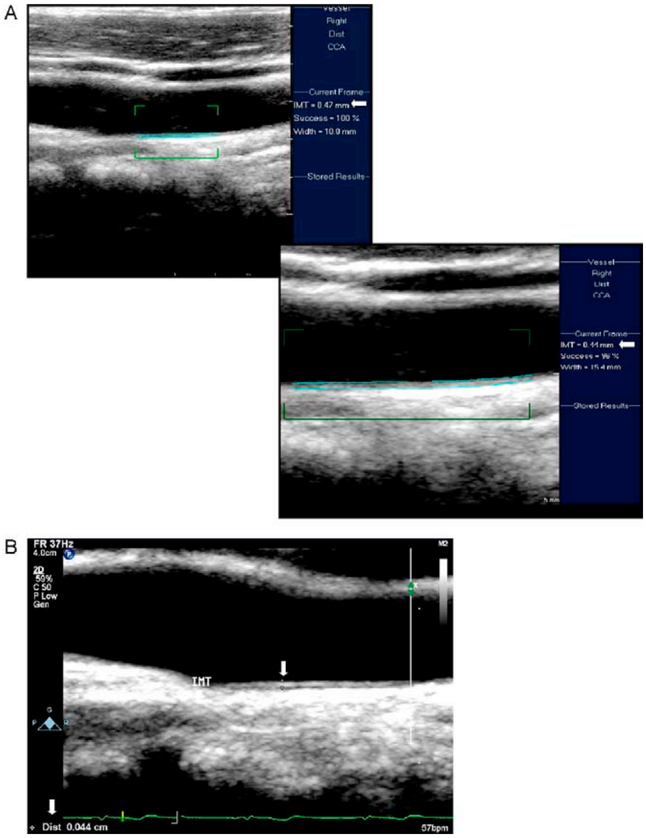

Figure 2 B-mode images of the carotid artery of the same patient. Comparative measurements of IMT: A and B — automatic measurements of mean IMT (0.47 mm, 0.44 mm), C — manual measurement of max IMT (0.44 mm). Compare to IMT measurement by M-mode (0.49 mm) from Fig. 1.

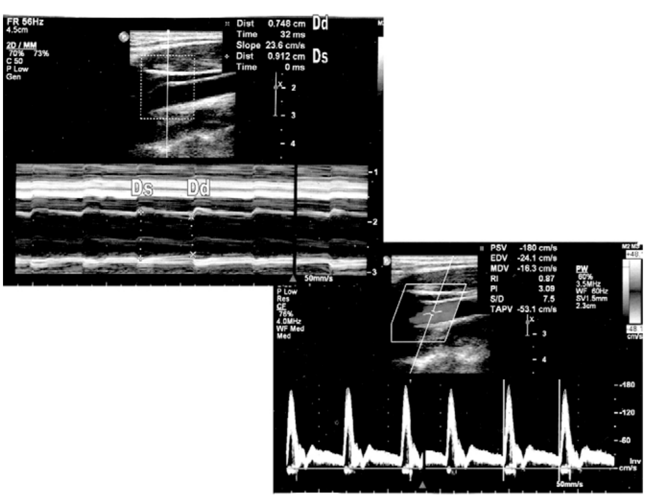

Figure 3 Representative M-mode (A) and color-coded duplex sonographic (B) images of carotid artery (near bulb region) of 18 years old female patient with low blood pressure (90/50 mmHg), and orthostatic intolerance. Increased artery distensibility during cardiac cycle Ds (marked as +) — Dd (marked as ×) = 0.16 cm. Significantly high peak systolic velocity —180 cm/s with diastolic drop: pronounced diastolic notch and significantly increased systolic/diastolic ratio 7.5.

carotid arteries but also in intracranial arteries were found in 48 of cases. 16 patients had additional mid-diastolic wave as additional wall distention (not exceeded 0.02 ± 0.01 cm) accompanied by high systolic blood flow velocity with a prominent increase of systolic/diastolic velocities ratio. All these patients had significant brachial pressure fall during orthostatic test indicated the lack of autonomic nervous regulation (Fig. 3).

Conclusion and discussion

The strong correlations exist between carotid arterial elastic properties and carotid baroreceptors, cardiovagal baroreflex sensitivity with an impact on arterial blood pressure and stroke volume. We can assume the interdependency between carotid distensibility and autonomic balance. Whereas some studies suggest that reduced elastic properties of carotid arteries cause the reduction of cardiovagal baroreflex sensitivity resulted in changes of hemodynamic, the results of our previous and recent studies show the dependence of carotid arterial distensibility on autonomic neural regulation of wall tone.

The autonomic imbalance in young people was associated with the increase of arterial distensibility, expressed as increase of carotid arterial systolic/diastolic diameter change, sometimes additional arterial mid-diastolic wall motion, accompanied by abnormal distribution of flow velocity during cardiac cycle with the marked systolic flow acceleration and significant increase of systolic/diastolic ratio.

This conclusion coincides with the findings of the decreased arterial stiffness in a young people under the acute sympathetic stimulation of artery. These results may be explained by an unloading of stiffer wall components during active arterial constriction under influence of autonomic stimulation [27]. The further comparable evaluation of patients with different impairment of nervous system could help to determine the role of nervous regulative function on arterial wall dynamics.

Taking into account many basic mechanisms and various factors influencing arterial wall dynamics it is difficult to measure the impact of each of them separately. Arterial wall stiffness or distensibility measurements reflect the dynamics of all structures of the arterial wall as well as dynamics of blood perfusion. Arterial mechanical properties can be calculated in different ways including parameters of various factors affecting wall motion. The development of the high resolution ultrasound tracking techniques makes it possible more accurate measurements of arterial elastic properties which is extremely important for early detection of vascular pathology.

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