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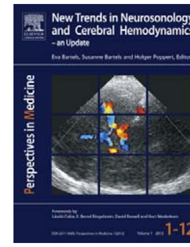

Arterial wall dynamics

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Distensibility;
Compliance;
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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:

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

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 $\mathrm{Ds} - \mathrm{Dd}$

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

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

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

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:

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

where SV is left ventricle stroke volume.

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

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

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:

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

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:

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

Pressure/strain elastic modulus (EM) is calculated as

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

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].

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

where IMT is intima–media thickness.

Stiffness index (β) is calculated as

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