FREQUENCE DISPERSION OF THE SURFACE WAVE - INITIAL FACTOR OF ATHEROSCLEROSIS

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ABSTRACT/INTRODUCTION: Separation zones and low/oscillatory shear stress play an important role in the development of arteriosclerosis. However, conventional researches of the pressure wave propagation, do not take into account the vessel wall and the viscoelastic properties of blood in conjunction with the wave reflection in resonance with the protodiastolic pressure alterations, despite it being important in the analysis of the shear stress.

BACKGROUND: Atherosclerosis is the leading cause of death in the developed world and nearly the leading cause of death in the developing world. Nonetheless, atherosclerosis remains a geometrically focal disease, preferentially affecting the outer edges of vessel bifurcations [1]. In these predisposed areas, hemodynamic shear stress, the frictional force acting on the endothelial cell surface as a result of blood flow, is weaker than in the protected regions [2.3].

Two contradictory hypotheses were advanced in the 1970s to explain this distribution of lesions [4]. The first implicated high shear stress via direct endothelial injury and denudation, as suggested by experimental exposure of endothelium to supraphysiological shear stress. The second invoked low shear stress.

Experimental and clinical observations have noted that early endothelial injury is more likely to occur in areas of blood flow separation and low shear stress. These areas of low shear force tend to occur at the outer walls of arterial branching points and have been shown to induce endothelial dysfunctions [5].

It has long been hypothesized that low wall shear stress and the resultant stagnation of blood permit increased uptake of atherogenic blood particles as a result of increased residence time. Prolonged oscillatory shear stress induces expression of endothelial leukocyte adhesion molecules, which are important in mediating leukocyte localization in the arterial wall [6].

THE AIM of the study is to investigate the arterial blood flow with the MRI and CT and identify the initial factors of atherosclerosis.

METHODS: At different sites of the aorta peak velocity, net flow, flow acceleration (Magnetic Resonance Angiography- flow quantification) and blood density (Computed Tomography - in Hounsfields) has been investigated in 17 healthy volunteers (18-52y).

RESULTS: At the outer curvature of aorta, in the end of the systole, flow separates. At the isthmus, flow acceleration at the start of the diastole is 11.6±0.6 times higher than that in systole. Net flow from systole to diastole increases 2.5±0.5 folds. From the end systole to the initial diastole there is plateau on the net flow graph: here, at the outer curvature of isthmus, group waves at the boundary reflection, change in phase at 180^0 at the sine wave oscillation frequencies -1.25Hz and 2.5Hz. Blood density, from the aortic isthmus to the abdominal aorta, equals to -51±3HU and - 31±4HU respectively.

Table 1. Flow velocity and acceleration in different sites of aorta.

Measurement site	Peak velocity (cm/s) in systole		Flow acceleration (cm/s^2) in systole (bold) and from the end systole to diastole (underline)	
	external curvature	inner curvature	external curvature	inner curvature
Entrance of the ascending aorta	80.3±2.1	71.7±1.7	422.7-445.4	378.4-396.7
Aortic arch	47.8±1.4	59.3±1.8	**227.0-265.9** 857.3-944.5	**310.8-330.3**
Isthmus area	60.1±1.2	74.7±2.5	**318.4-331.4** 3657,2-3865.3	**390.3-417.3** 571.4-685.7
Thoracic aorta	62.6±2.4	59.9±2.7	**325.4-351.4** 330.3-354.7	**309.2-338.4**

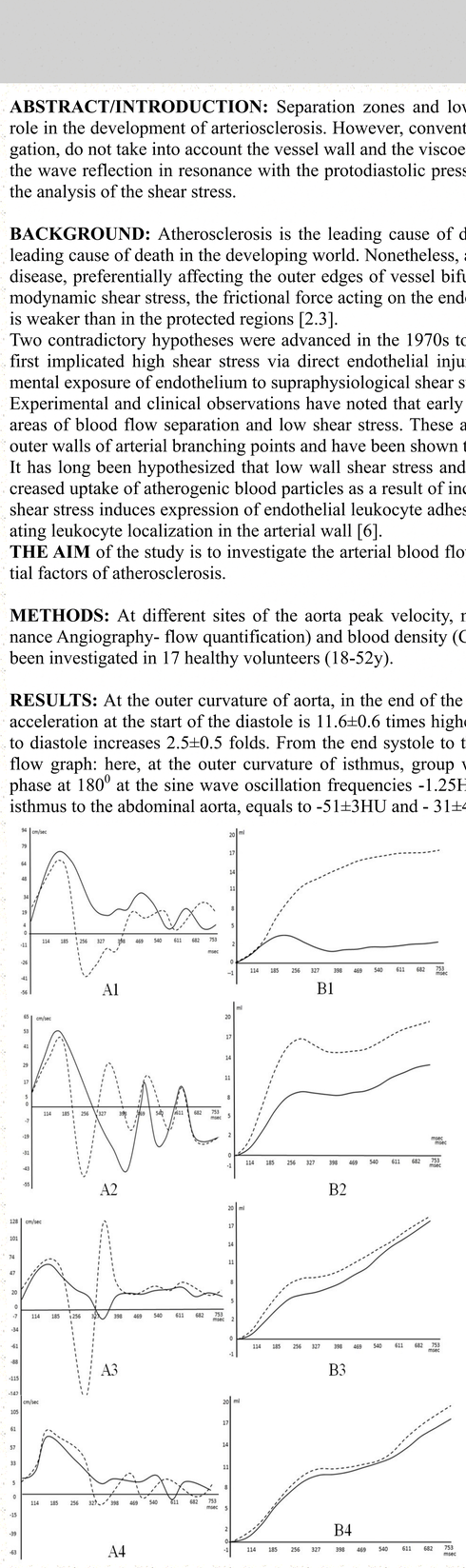


Figure 1. Peak blood flow velocity A, and net flow B. at the: 1-ascending aorta, 2-Aortic arch 3. Isthmus of aorta, 4. Thoracic aorta.
At the ascending aorta (1). (Dotted line — flow at the outer curvature).
velocity of blood flow at the outer curvature is lower than the velocity at the inner curvature.
At the peak velocity graph separated streams flows in the opposite directions.
Net flow increases at the end of the diastole.
At the aortic arch (2) on the outer curvature forward wave spreads to the periphery, where it does not dissipate, but is reflected back to central vascular system. Standing waves arise.
Flow direction alteration happens many times.
The resulting waveform is determined by the phase sum of forward and reflected waves.
Wave envelope forms the group wave nodule at the 300 msec. On the net flow graph from the end of systole to the initial part of diastole there is a plateau.
At the isthmus of the aorta (3) from the 185msec, blood flow separates into oppositely directed streams resulting in the wave superposition. The group wave at 327 msec. forms the wave nodule. Zero flow velocity is specified to the whole volume in the single slice. During the initial diastole, peak velocity sharply increases at the outer curvature and becomes higher than in systole. Net flow increases at the end of the diastole, while from the end of systole to the start of diastole there is a plateau.

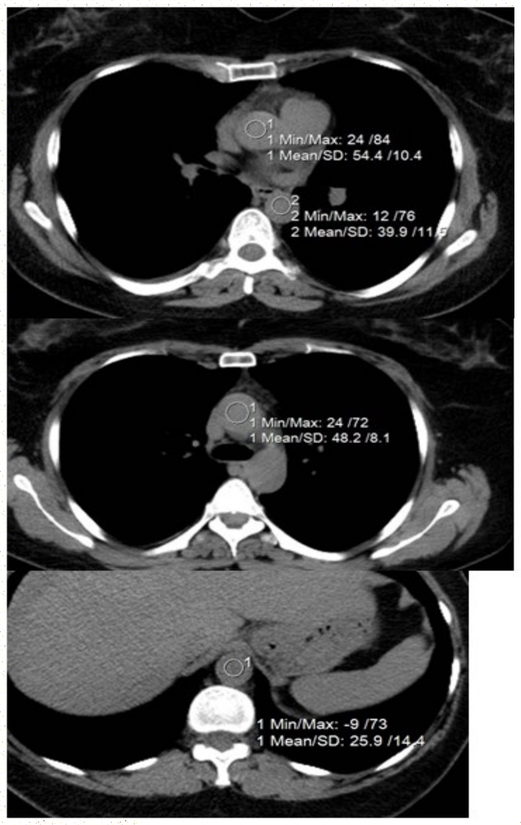
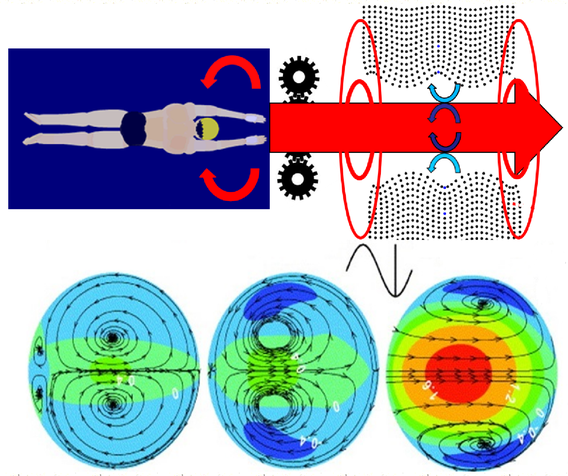


Figure 2. Density (HU) of blood decreases to the distal of aorta (entropy of the blood increases)



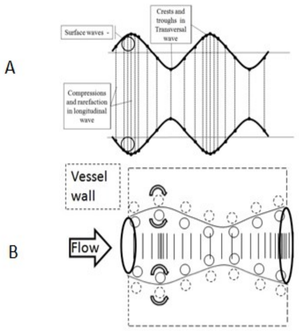


Figure 3. (a) Correlation between transverse and longitudinal waves. A transverse wave is made up of crests and troughs while a longitudinal wave is made up of compressions and rarefaction. (b) Surface wave formation on the boundary layer and the adjacent vessel wall.

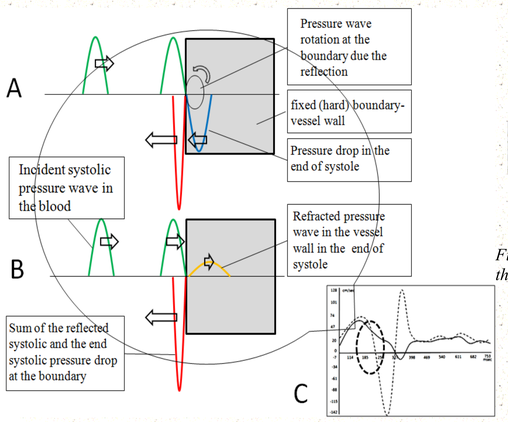


Figure 4. Surface wave rotation during the reflection. (A) If the incident medium has a lower index of refraction then the reflected wave, there is 180˚ phase shift upon reflection. On the outer wall of the aortic isthmus, reflected wave is in phase with the pressure drop (180˚ phase delay from the initial systole) during the end of systole, giving the total pressure sum with the amplitude twice as great as near the surface.(B) Reflected and refracted/transmitted waves have different frequency, velocity and direction. (C) Analysis made from the MRA- blood flow.

ADVANCES IN KNOWLEDGE:

Fundamental mechanism of the atherosclerosis.

Role of the pulsating motion in blood flow.

Confirmation of the blood and vessel wall viscoelastic alterations during the motion on the boundary layer.

Modification of the group wave, is the basis for the recovery of the vessel wall in atherosclerosis.

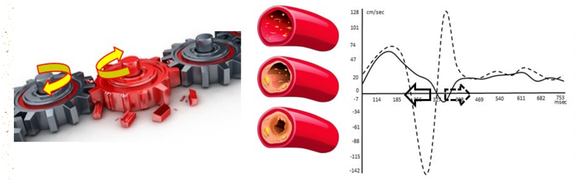


Figure 5. Gear is damaged, when the motion of cogs are opposite *Atherosclerotic plaque* *Blood flow at the isthmus of aortic arch. Opposite streams of flow in the initial diastole.*

1. *In protodiastole blood flow is bi-directional with the different frequencies of oscillations in distinct streams.*
2. *On the boundary layer it forms dispersion of frequency between the flowing substance and the vessel wall. This is the initial factor of atherosclerosis.*
3. *Separation of flow is created from the external wall of the branching sites, where waves directed oppositely create superposition.*

CONCLUSION: Pulse pressure propagates through the blood and the vessel wall. On the boundary layer it forms surface wave. Blood is viscoelastic substance. Pulse pressure, during reflection at the outer wall of aortic isthmus and at all arterial branching sites, is in resonance with the end systolic pressure drop. Amplitude of the stress, associated with the vessel wall, increases. Waves with different frequencies are formed at the end of systole, at the outer wall of the circular flow sites. At the same time flow separates. Wave packets at the frequency dispersion, destroys the cell aggregates increasing the entropy of blood, whereas at the boundary layer of the vessel, it denudates endothelial sheet.

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