# Frequency dispersion of the pressure wave on the vessel wall - primary reason of atherosclerosis.

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**Introduction:**
Blood flow-induced shear stress now is accepted as an essential feature of atherogenesis.
Here we present blood flow conditions at the different sites of aorta and investigate stress due to flow.
Compared to existing solutions [1] we give a) theoretical basis [2.3] for the instantaneous highest shear stress formation at the end systole at the circle flow sites (due to pressure reflection and superposition - resonance);

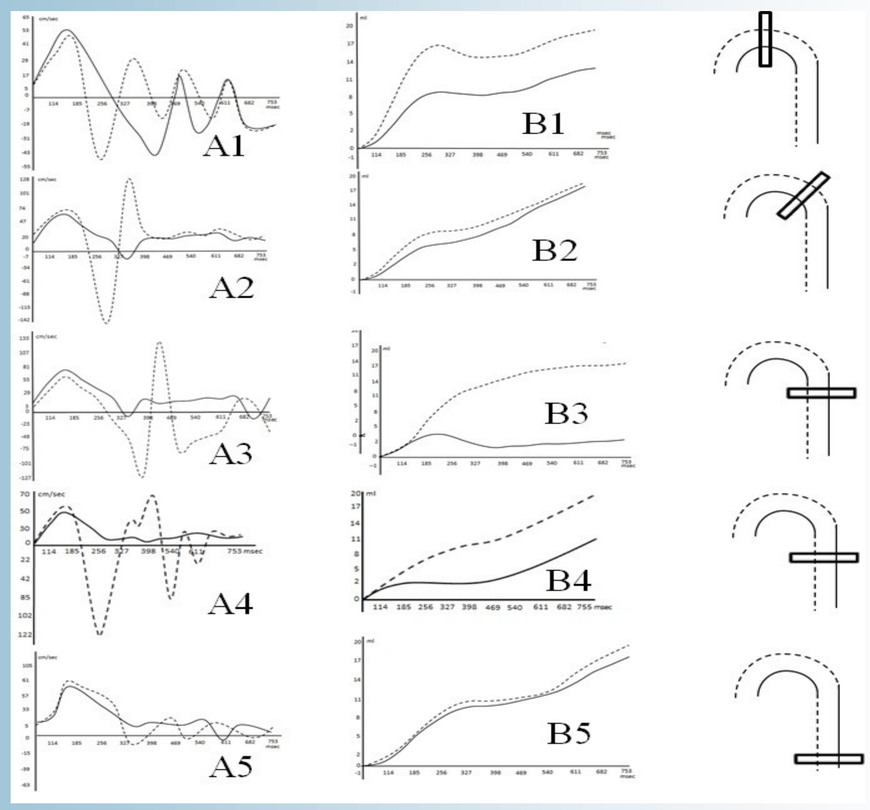


**Fig1**. Peak blood flow velocity (A) and net flow (B). (Dotted line - flow with the high shear stress at the reflection).

Blood flow velocity at the outer curvature of the aortic arch (1) is lower, than that at the inner. Net flow increases at the end diastole. In the aortic arch standing waves arise. Flow direction alteration is in many times. The resulting waveform is determines by the phase sum of forward and reflected waves. Wave envelope forms the group wave.

Isthmus of the aorta (2) from the 185msec, blood flow separates into the opposite directed streams resulting in the wave superposition. The group wave at 327msec. forms the wave nodule. Zero flow velocity is specified to the whole volume in the single slice. In the initial diastole peak velocity sharply increases at the outer curvature and is higher than that in systole. Net flow increases at the end diastole.

Upper thoracic aorta (3). Low thoracic aorta. Reflection from the inner wall (4). Wave at 327msec. forms the nodule. Flow at the different walls equalizes (5).

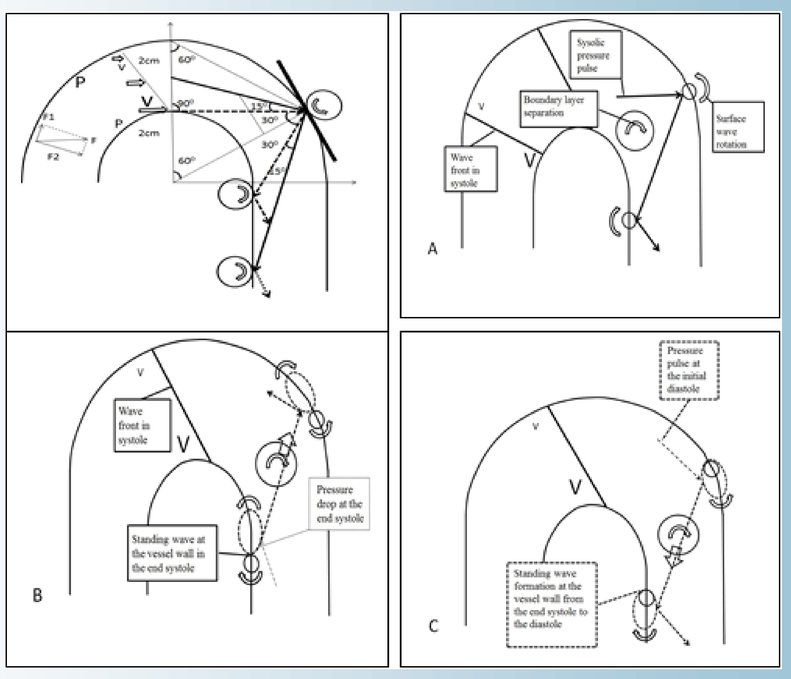


**Fig2.** Pressure and flow velocity distribution at the aortic arch in systole (left-above). Standing wave formation due to resonance at the outer wall of the isthmus and inner wall of the thoracic aorta: a.peak systole, b. end systole, c. initial diastole (right). Standing wave frequences and resonance oscillation of the first harmonic (a), and the second harmonic (b,c). Vortices at the flow separation, are displaced to the vessel wall. At the external wall of isthmus rotational kinetic energy adds to translational kinetic energy and pressure increases.

**Results:**
At the outer curvature of the aorta in the end systole flow separates. At the isthmus, flow acceleration in the initial diastole is 11.9 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 a plateau on the net flow graph. At the outer curvature of isthmus, group waves at the boundary reflection, changes in phase at $180^0$ at the oscillation - 0.8Hz and 1.6Hz. Blood density from the aortic isthmus, to the abdominal aorta equals to - 51±3H to 31± 4H respectively.

**Conclusion:** Blood motion at the boundary layer, due to blood and vessel wall viscoelasticity, forms the surface wave. At the outer wall isthmus of the aorta, pulse pressure at the reflection is in resonance with the end systolic pressure drop and amplitude of the wall stress increases. At the end systole wave packets with the different frequencies are formed. Erythrocyte aggregation at the pulse ejection, forms the high elastic load, and energy, without loss, transmits from the heart to the blood and all the human cells . Frequency dispersion destroy the cell aggregates in the flow stream, whereas at the reflection and cyclic load of the wall, shears the endothelial sheet.

[1] Kristopher S Cunningham, Avrum I Gotlieb. Lab Investigation (2005) **85**, 9–23.

[2] Daid Halliday, Robert Resnick, Jearl Walker. Fundamentals of Physics. John Wiley & Sons, 2011.1328p.

[3] Julian F. V. Vincent. Structural Biomaterials. Princeton University Press, 1990. Medical. 244p.