

What is the “physics” of the process of spasm of large arteries and atherosclerosis?

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Goal

It is known that a person's life expectancy depends on the degree of acquired atherosclerosis. But doctors do not yet know what the mechanism of atherosclerosis development is. Researchers refer to 30 risk factors, mainly related to the lifestyle of a modern person. But how do these risk factors turn normal elastic artery walls into harder and plaque-covered walls by the age of 30-40? That's the question.

Method

The study of numerous sources of information posted on the Internet. Discussion of proposed ideas at conferences and webinars, publication of original articles in Russian and English-language medical journals [1-5].

Results

Gradually, over more than 100 years, researchers have come to a consensus that it is the endothelium that is the main link in the pathogenesis of atherosclerosis. Yes, it is endothelial dysfunction that leads to atherosclerosis of the arteries and, further, to other cardiovascular diseases. But what force or what process is the root cause of endothelial damage and the development of atherosclerosis was not answered until 2020. Most scientists have delved into the wilds of genetic engineering, into the biochemistry of processes in atherosclerosis. The results are not impressive yet. The author of the New Theory showed that such a force is a physical force that acts on the endothelium and muscle layer of the walls of such vessels from inside the arteries. The author of this article managed to answer two main questions facing modern cardiology.

1) Why do atherosclerosis and some other CVD begin with endothelial dysfunction?

2) Why are low-density lipoproteins (LDL) the main “culprit” of atherosclerosis?

The answer to the 1st question. Frequent stress is accompanied by an increase in blood pressure, the opening of anastomoses and the leakage of arterial blood from the arteries into the veins. Due to the lack of arterial blood, there is a decrease in the volume of the arterial bed, spasm of the walls of the arteries. The spasm is forced. The area of the lining of the arteries, that is, the area of their inner surface, decreases, because at moments when a person is nervous, the volume of arterial blood decreases. The aorta and arteries closest to the heart and slightly higher are particularly susceptible to spasm. In particular, the unicellular layer of the endothelium is compressed, the distance between the cells is reduced to a minimum. The surface of the endothelium contracts laterally along the “circle”, the lumen of the artery decreases, the inner layers thicken, undergoes shifts, changes up to the separation of part of the cells, “holes” form in the unicellular layer of the endothelium of the arteries and aorta. Such mechanical influences eventually lead to an increase in the rigidity of the walls, to a decrease in the production of NO in the damaged walls, which accelerates the “aging” of the arteries.

The answer to the 2nd question. With arterial spasm, “suction forces” arise in the multilayer walls of the arteries stretched inward relative to the incompressible adventitia. This is due to stretching of the middle muscle layer of the arteries, as well as due to an increase in the volume of the muscle layer, since the adventitial layer practically does not change its size, and the internal endothelial is compressed, reducing the lumen of the arteries. Negative pressure in the middle layer due to individual endothelial damage draws the lightest fractions of blood into the muscle layer first of all. The easiest are LDL, so basically they are forced to penetrate and “seal” the damage in the endothelium. The physical process of “suction” is the reason for the movement of LDL into the depth of the artery wall. But these different fractions of blood are foreign to the walls of the arteries, they should not be there in such quantity, so the arteries undergo a natural inflammatory reaction, the formation of plaques.

The author believes that physical exertion, special breathing exercises, blood transfusions into the arteries are the salvation and prevention of atherosclerosis, because in order for the arteries to remain healthy longer, it is necessary to periodically and prophylactically replenish the volume of arterial blood.

Conclusion

It is possible to increase blood flow to the arteries by strengthening the function of the pulmonary arteries, that is, due to the additional work of the small circle of blood circulation. Such procedures as singing, laughing, “playing

the flute", therapy with powerful screams (at football) are useful. Periodic replenishment of arterial blood volumes is the main thing that keeps the cardiovascular system from atherosclerosis, from aging. As follows from this theory, LDL is not to blame for anything, external physical force drives LDL molecules under the endothelial layer and causes atherosclerosis!

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