

Possible Cause of Atherosclerosis from the Point of View of a Physicist

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Abstract

It is believed that at the moment the cause and mechanism of atherosclerosis are still unknown. Only a large list of related factors is known. More than 50% of people die from the effects of atherosclerosis. The author of the New Theory of CVD in 2020 made an attempt to show a completely new idea of its origin and development, to answer the question why atherosclerosis is almost inevitable for every person. Perhaps this is a sensation, but none of the cardiology schools is in a hurry to admit, or at least openly discuss a new hypothesis. By the beginning of 2021, several author's articles on the causes of atherosclerosis were published [1,2].

Keyword: *Atherosclerosis; Physicist; CVD*

Purpose of the Study

The purpose of this article is to draw additional attention to this problem of humanity, because not every year something new is proposed in cardiology.

Methods

Study of information on the Internet, participation in medical conferences, publication of articles.

Results

Atherosclerosis, as the main disease, has attracted the attention of medical scientists in many countries only in the last 120 - 150 years.

Since the end of the 19th century and until now, a huge amount of effort and money has been spent on research into the causes of atherosclerosis in the world, but the mystery has not been revealed. Many studies, mostly useful, have been conducted around this problem, but plausible causes and mechanisms have not been found. Research continues.

Doctors are constantly studying the spread of atherosclerosis in different geographical areas and in different population groups. As a result, it was found that the degree of vascular damage depends on climatic and geographical conditions, nutrition, professional activity, housing conditions, age and character of a person. Vascular changes caused by atherosclerosis are found in almost all people, more often from the age of 30 and mainly in men. However, doctors say that atherosclerotic changes in themselves do not determine much. The famous Russian scientist I. V. Davydovsky claimed (1966) that atherosclerosis is not a disease, but a natural biological process.

But what about the immutable fact that atherosclerosis of the heart vessels is a favorable ground for the development of coronary insufficiency and myocardial infarction? Here the cardinal question arises: why in some cases we are dealing with a disease and why in others, exactly the same changes do not entail any consequences? Or why does a certain degree of atherosclerosis of the vessels that feed the brain sometimes lead to strokes or ischemic attacks, and sometimes not?

Come on, all in order. Let's start with an elusive parameter for modern medicine: the optimal volume of arterial blood for a healthy person of a certain height and optimal weight? How important is the optimal arterial blood volume? What should be the ratio of arterial (A) and venous (B) blood volumes? Are these parameters important?

It is necessary to study the variability of these parameters. It is known that the volume of arterial blood [3] in a healthy person $A = 15 - 20\%$, the volume of venous blood together with capillary $B = 80 - 85\%$. Let's take $A = 17.5\%$, $B = 82.5\%$, the volume ratio is conveniently taken as $A:B = 100: 471$. In other words, venous blood is 4.71 times more than arterial blood, so in the process of blood circulation, on average, the exchange of the entire volume of blood in the veins is 4.71 times slower than in the arteries.

According to the New Theory of CVD, one of the most important conclusions is that physical or psychological stress and some other causes lead to an increase in blood pressure (BP), and an increase in blood pressure leads to the opening of large arteriovenous anastomoses of the ABA, for example, between the mesenteric artery and the portal vein [4]. The pressure in the arteries is sharply reduced. Permanent and prolonged loss of arterial blood through the ABA can lead to a temporary decrease in the internal volume of the arterial bed and to an increase in the volume of venous blood. A sign of a lack of arterial blood: at some point, the hands and feet become cold. We emphasize that the volume of arterial blood and the internal volume of the elastic arterial bed are equal at any time. A decrease in the volume of arterial blood inevitably leads to a decrease in the internal volume of the arterial bed. In this case, the four-layer walls of the arteries, consisting of a rigid adventitia, elastic media, thin intima and single-celled endothelium, are forced to contract, each layer is compressed by its own value, as a result, the internal lumen of the arteries decreases, while the nutrition of the "vessels of the vessels" deteriorates. The endothelium and intima are damaged, because when the internal diameter of the vessel decreases, the endothelial cells shift and "crawl" on top of each other. "Gaps" are formed, through which cholesterol molecules penetrate under the intima and "seal" the gaps.

Note that the heart pulse itself also compresses and decompresses the walls of the arteries: 60 - 80 times per minute, but it is important to note that in a young and healthy person, the pulsation of the walls occurs around the average equilibrium diameter of the arteries. Such constant fluctuations of the walls of the arteries can be called continuous massage of the arteries, contributing to normal both general blood circulation and blood circulation of the "vessels of the vessels".

Well, if some of the blood has flowed into the veins, then the transverse vibrations of the arteries do not occur around the equilibrium state of the arteries, but around the tense (spasmodic) state of the arteries: the walls are compressed in the transverse direction, while the rigid adventitia keeps the diameter of the outer layer of the arteries as much as possible, the elastic middle layer (media) is maximally stretched towards the axis of the vessel, the intima and endothelium are affected by the forces of separation from the media, also towards the center of the vessel. According to the laws of physics, intima is most often damaged in the areas of arterial bends and bifurcations. Due to compression, the diameters of the layers decrease, the smooth muscle cells from the media become "cramped" inside their layer, so during spasms, there are migrations of "extra" cells into the intima. The intima thickens, the stiffness of the walls increases, plaques grow.

It is clear that the greater the leakage of arterial blood and the longer the lack of arterial blood is retained, the greater the stretching of all layers towards the center of the vessels, the longer the forces of separation of intima from the media act.

Note that under normal conditions, the throughput capacities of the left and right ventricles are the same, so increasing pumping in a small circle with passive human behavior is difficult.

At this point in the story, for a better understanding of the subtle mechanism, it is logical to give an example from physics. If you pour water into a long test tube (here the test tube plays the role of an aorta or a large artery, the statics of the process are considered, but the dynamics are not considered), then quickly turn the test tube over and lower the open end a few cm into a basin of water (analogous to a venous pool), then the water from the test tube will not pour out, or rather, only a very small part will pour out. Similarly, only a small part of the blood flows from the arteries to the veins when the ABA is open. What forces will act on the walls of the test tube from the inside? Exactly the same as the forces of the vertical column of arterial blood on the internal walls of the arteries, and in the upper parts of the arteries, the separation forces are more powerful, but the glass in the test tube is almost incompressible, so we do not see a decrease in the diameter of the test tube. But if the test tube is made of soft and fairly thin rubber, then we will see the effect of compression of the vessel. And the more elastic the rubber, the greater the compression and deformation of the vessel, especially in its upper part!

We can conclude that if the spine and aorta are in an upright position for a long time (during the day), then all this time there is a force of stretching the media in the transverse direction (towards the center of the vessel) and the force of separation of the intima and endothelium from the media. But at night, in the horizontal position of the body, the walls of the vessels get rid of the forces of separation of the inner layers from the outer ones, and the arteries get some time for relaxation, recovery. When a person has frequent stressful situations with loss of arterial blood through the ABA and the restoration of arterial blood volume is delayed, then destructive events in the aorta and arteries increase and accumulate.

We emphasize here that both the test tube and the human arterial system represented in the physical experiment are affected by similar forces generated by fluids. These forces do not act from outside, but from inside the vessels. Direction of forces: to the axis of the vessels, in the extreme case, the walls of the vessels can close. Moreover, we know from hydraulics that in a tube with a variable diameter, in those places where the diameter decreases, the velocity of the liquid increases proportionally and its pressure on the walls decreases. This means that the elastic tube will have a tendency to sharply reduce the lumen of the vessel in the area of the plaque, up to complete obstruction. The result may be as follows: an acute attack of angina, in fact, a "spasm" of the aorta or large arteries. When the ABA is located between the mesenteric artery and the portal vein, it can be the arteries closest to the ABA: coronary or cerebral. End result: significant ischemia and possibly a heart attack or stroke.

Of course, in the body of a healthy person, automatic adjustment of blood volumes is provided, the volume of arterial blood is replenished to the optimal value, or to the ratio of arterial and venous blood volumes in the proportion $A:B = 100:471$. In a person with heart failure, with venous fullness, the venous part of the blood increases significantly, for example, to $B = 471 \dots 600$. Replenishment of the arterial blood volume can occur only by increasing the throughput capacity in the small circle of blood circulation. You can help the process through physical activity and/or increased breathing exercises. In children and young people, the replenishment of the arterial pool can occur naturally: they are engaged in physical education, play, and constantly move.

But the regularization of blood volumes can still be disrupted by the age of 30. This happens with inactivity, with prolonged overeating and weight gain, with hunger or poor nutrition, with adversity, when living and working in cold climates, in cold apartments, with constant stress. The mechanisms of influence on the degree of atherosclerosis have yet to be studied. But it is clear that with age, the degree of atherosclerosis usually increases for two reasons. First, because of the constant influence of the Earth's gravity on vertically positioned vessels, this influence lasts for a lifetime. (Compare the degree of atherosclerosis in animals whose spine is located horizontally-usually there is no atherosclerosis!) Secondly, due to nervousness, there is an additional narrowing of the arteries due to periodic losses of arterial blood.

In the opposite direction, atherosclerosis itself does not go, because the body cannot quickly make up for the loss of arterial blood volume and increase the average diameter of the arteries [5]. In reality, the body can only adapt.

On the other hand, in order for the body to counteract further leaks of arterial blood, spasm of the main arteries, dissection of the aorta and large arteries, the walls of the arteries are strengthened, while reducing their diameter - this is the natural cause of atherosclerosis

and the growth of plaques on the walls of the arteries as the age of a person increases [6,7]. Gradually, the elastic arterial bed is replaced by a bed with more rigid tubes, so in accordance with the stiffness of the walls, the cardiac output also decreases, since the power of the left ventricle becomes insufficient to create a normal blood flow through the rigid aorta and arteries. Cholesterol, calcium, and other elements are just improvised building materials for fixing walls and “treating” damage. After all, if atherosclerosis did not form and the walls of the arteries would continue to remain soft and pliable, then very quickly general ischemia of most organs would occur, and a person would die earlier. Apparently, for modern people, throughout his history leading a life in conditions of numerous hardships and adversities, atherosclerosis has a positive meaning, it prolonged a short life. Data on how the prevalence of atherosclerosis increases during the war due to nervous stress are given in the sources [8,9].

So, the constant action of gravity and the constant periodic decrease in the volume of arterial blood over the years, resulting in an increase in the stiffness of the walls of the arteries and a decrease in the release of blood by the ventricles, leads to ischemia of the heart, brain and other organs.

Apparently, in order to slow down the rate of decrease in the volume of the arterial bed, the development of atherosclerosis and the growth of plaques, it is necessary to periodically transfuse your own venous blood into the arteries. But this procedure is still hypothetical, requiring proof and confirmation.

In addition, to combat stress and increase blood pressure, you need a proper lifestyle, a varied diet, fighting excess weight, daily physical activity, and breathing practices. Periodic cleansing of the body is also useful, for example, by the hijam method [10] and others.

Conclusion

Thanks to the New Theory of CVD, the mechanism of the formation of atherosclerosis in an erect person in the conditions of earth gravity is shown. In essence, the degree of atherosclerosis is proportional to the degree of periodic loss of arterial blood (its overflow through the ABA into the venous pool), forced reduction of the arterial bed (spasm of the artery walls), multiplied by the time of such imbalances. The adaptation of the body is activated. The only correct solution for adapting the cardiovascular system is to increase the stiffness of the arteries. It becomes clear that the optimal balance of arterial and venous blood volumes should be close to the ratio $A:B = 100:471$ and it should be adhered to.

At the same time, the mechanism of aortic dissection is shown: due to the forces of separation of the inner layer of the vessel wall in the vertical column of blood, especially in its upper part.

Apparently, during the life of a modern person, the parameter A can decrease to 80...100 and the parameter B very often, on the contrary, increases to 471...600 and higher. In the future, in order to maintain the elasticity of the arteries, a person will have to learn, with the help of medical procedures and devices (which still need to be invented), to maintain a constant volume of arterial blood and the balance of arterial and venous blood volumes at optimal levels for as long as possible.

There is a lot of work to be done on the actual fight against atherosclerosis. Namely, the work is waiting for doctors, physicists, microbiologists and other specialists.

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