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Short Communication

Once again about the main cause of atherosclerosis

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Abstract

Annotation. The article is devoted to the most important topic in medicine: the causes of atherosclerosis. It seems that over the past 200 years, thousands of prominent scientists have approached this problem from different angles, and by today all ideas for possible causes have already been exhausted. But no, in 2020 another idea was found that is amazing in its simplicity and effectiveness, like a chess checkmate in 3 moves!

Keywords: causes and mechanism of atherosclerosis; arteriovenous anastomoses; arterial blood volume;arterial spasm

Goal. The cause of atherosclerosis is the main problem that modern medicine has not been able to solve for more than 100 years. Let's discuss a few facts that will show that since 2020, the new mechanism of atherosclerosis has finally begun to become clearer and in the future this mechanism will apparently take hold of the masses.

Methods. Searching for information in the literature, participating in conferences and discussions with leading Russian cardiologists.

Results.

Modern medicine says that a person may have several causes of atherosclerosis, and lists a long list of them. But the list presented is a list of factors that influence only the rate of development of atherosclerosis. It is known that there must be one true, dominant cause for any phenomenon! Thus, the mechanism of atherosclerosis must be one, influencing in such a way that the development of atherosclerosis occurs in different people at different rates! But if there are currently more than a dozen declared causes, then it is necessary to conduct an "audit" of all the "evidence" and look for the main, perhaps unknown, reason for the development of atherosclerosis.

Fortunately, such a reason has already been found [1] in 2020! But academic circles are in no hurry to recognize it, apparently, it is to someone's benefit to remain ignorant. I note that so far no one has written me a negative review of the new hypothesis, although for 4 years it has been widespread not only among Russian-speaking, but also among English-speaking readers [2]. We can assume that the silence of the touchy academicians is confirmation of the new hypothesis!

So, as before, the main link in atherosclerosis and many cardiovascular diseases (CVD) is periodic stress. Previously, it was assumed that stress acts directly through the central nervous system (CNS) to destroy the cardiovascular system.

The new idea says: yes, stress is the main cause of atherosclerosis and other cardiovascular diseases. But it is impossible to get rid of stress. That's life!

According to the author, it is necessary to find what follows stress. It turned out that stress can lead to periodic loss of some volume of arterial blood. How? When blood pressure (BP) increases under stress, arteriovenous anastomoses can open, transfer blood directly into the veins, and then close [3].

Let's look at the work of the cardiovascular system, but from the other side. A person has a large systemic circulation (SC) and a pulmonary circulation (PC). It is known that each cell of the body must be continuously supplied with nutrition and oxygen through capillaries. The total volume of arterial and venous blood is about 5000-5500 ml. The main link in the supply of all organs is the arterial reservoir with a volume of Vb, filled with arterial blood with a volume of Vk. These volumes, if we take into account the volumes of all arteries above the arterioles, are always equal to each other, i.e. Vb=Vk, they cannot be unequal, because plasma is inextensible and incompressible. But the walls of the arteries are tensile: both along and across the axes of the vessels. Muscular-type vessels have to adapt to the volume of blood in the arteries and, when there is a shortage of blood, "shrink and strengthen," while losing their elasticity!

Strictly speaking, the total volume of arterial blood changes slightly with each heartbeat. This volume is replenished and emptied by approximately the same amount as the volume of cardiac output of the systemic circuit and blood flow in the general capillary bed. Normal cardiac output is 70-100 ml, the average volume of arterial blood in a person is about 850-950

ml. Thus, with each cardiac output in the total volume of arterial blood Vk, blood is replaced by 7-10%. This is a very large quantity! Indeed, on average, in one minute at a heart rate (HR) of 70 beats, the entire volume of arterial blood passing through the upper part of the aorta and large arteries manages to be replaced with new blood about 7 times!

To deliver nutrition to each working cell, the required blood pressure (BP) is maintained in the arterial basin. On average, a person's blood pressure is 120/80 mm Hg. Depending on stress or physical activity, blood pressure may increase or decrease adequately. To minimize energy losses when pumping blood, the arterial pool has smooth elastic multilayer walls. The walls of large arteries always pulsate noticeably. The main thing is to understand that the arterial pool is a set of elastic "tubes" connected into one whole, of different diameters: the pool begins on the outside of the aortic valve, includes the largest artery called the aorta, then branches into large and smaller ones arteries. The bed ends with arterioles and millions of capillaries. The pressure along the entire length from the aorta to the arterioles and further to the capillaries gradually decreases from a value of 120/80 to 30...15 mm Hg. The cross-sectional area of the aorta is about 8 cm2, and the total area of all capillaries is 3200 cm2. The cross-sectional area of all capillaries is approximately 400 times larger than the area of the aorta, which is why the blood flow speed drops from 20 cm/sec at the beginning of the aorta to 0.05 cm/sec in the capillaries.

If you mentally pick up any "tube" (artery) of medium diameter filled with liquid, you can find out the following. The "tube" is characterized by elastic, non-collapsing walls, the cross-section of the "tube" is capable of maintaining a rounded shape and its diameter, and any segment of the "tube" is able to maintain its internal volume.

The total volume of the arterial pool Vb and the volume of arterial blood Vk are always approximately equal, because the ejected volumes of pumped blood in a healthy person through the left and right ventricles are equal (Harvey's law), and the venous volumes of returned blood from both circulation circles are also equal. In addition, under normal conditions in a healthy person, pulsating changes in the walls of the arteries are close to their average values, mechanical tensile stresses and compressive stresses in the walls change cyclically around their average zero values

The human vascular system contains large and small arteriovenous anastomoses [2], some of which are, in fact, emergency valves for reducing blood pressure in a certain area of the arterial basin. These valves open to urgently reduce pressure, while arterial blood is transfused directly into the venous volume, blood pressure decreases, and the valve closes. On the other hand, at the same time, the volume of the venous pool of the SC increases by the amount of overflow. Note that this venous pool has the ability to significantly expand (or decrease) without significant consequences. And now about the main thing.

A person who works, for example, at a computer or sits for a long time in front of the TV, during periods of stressful nervous loads, significant leaks of arterial blood occur. At high blood pressure, blood can flow into the veins due to the opening of large anastomoses.

Heartbeats create alternating stresses in the walls of the arteries. The processes in the cardio vascular system can be divided into two processes, which are carried out by static and dynamic forces.

The pulsation of blood vessels and the dynamics of blood flow in all parts of the arterial bed are not considered in detail in this article. It is known that in hydrodynamics, for some approximate estimates of stresses in the walls of pipelines, such simplifications are permissible.

In this article we consider this process only from the static side.

As a result of arterial blood leaks through the anastomoses, there is a decrease, at least for several minutes or even hours, in the volume of arterial blood Vk inside the fixed volume of the elastic arterial bed (pool) Vb. How do arterial walls react to loss of blood volume?

The pulsating walls of the arteries begin to fluctuate not around their optimal sizes, but around sizes reduced due to lack of blood volume, while the mechanical loads on the arteries increase. The walls of the arterial bed on its inner side experience a shear load inside the vessel, namely, the stress of separation of endothelial cells from the muscle layer increases towards the axis of the artery. On the other hand, the hard outer layer of the artery walls, i.e. adventitia layer retains its original size. As a result, there is a forced decrease in the internal diameter (lumen) of the artery due to transverse stretching of the muscle layer inward, which means that at the same time there is a decrease in the area of the lining, endothelial layer area. Endothelial cells become "crowded" on the inner surface of the artery

Let's evaluate how "crowded" the cells of the endothelial unicellular layer are. Thus, if a coronary artery, say, with a nominal diameter of 5 mm, at a certain period of time experiences a stretch on average of 0.5 mm due to the muscle layer (i.e., the internal diameter of the artery lumen decreases to 4 mm), then the area of the lining endothelial layer will decrease by as much as 20%!This can lead to alteration and dysfunction of the endothelium, and to the detachment of individual endothelial cells. This is how arterial damage occurs. And such events during stress can happen "unnoticed" with varying intensity every day.

What else should happen when the internal diameter of the arteries decreases? It is clear that negative pressure is created inside the transversely stretched muscle layer of the artery walls. In other words, plasma leaks primarily from the main flow deep into the arterial walls. Any very small fractions of blood, mainly LDL, can, due to the action of suction forces, penetrate through the cracks and holes of the single-cell endothelium into the walls of the arteries and "fix" there, destroying the tissue of the muscle layer. This may be the cause of persistent micro-inflammation of the arterial walls with an increase in the level of C-reactive protein in the plasma [4]. And such violations can occur for months or even years. Heart failure gradually develops, because increasing aortic stiffness leads to decreased cardiac output. The body's reaction: growth of plaques, further increase in the rigidity of the walls of the aorta and large arteries, i.e. progression of atherosclerosis. At the same time, heart failure increases, and overall health deteriorates. Where can plaques and arterial wall alterations appear first?

Firstly, where conditions are created for increasing mechanical stress in the walls of the arteries. According to calculations using hydrodynamics, in places of narrowing or expansion of a vessel, in places of branching or bending of vessels. These places are near or above the heart.

It turns out that if a plaque has appeared, then it is forever, because the plaque protruding into the artery itself becomes the site of additional narrowing of the vessel. At this point, the speed of blood flow increases, and the relative pressure above the plaque decreases, which promotes additional growth of the plaque, because sharp changes in blood pressure are created from high to low, and back! It is impossible to "cure" a plaque,

you can only install a stent, but then the same physical suction forces will contribute to the development of a new plaque in the vicinity of the stent.

Secondly, the influence of the volume of arterial blood leaks must be taken into account. Where in the arterial bed will this deficiency affect the most? It is reasonable to assume that in the upper region of the largest vessel. It is in the aorta, on the outer side of the aortic valve, that extreme negative pressures and corresponding stresses in the walls of blood vessels can arise there during diastole. In addition, due to the influence of the Earth's gravity, it is there that additional maximum stresses and spasms of the vertically located aorta are created. The areas most affected by negative pressure are the aortic valve, the upper part of the aorta, and the first arteries leaving the aorta. Oddly enough, the names of these first most vulnerable arteries are the right and left coronary arteries. Apparently, it is no coincidence that most often it is these arteries that begin to signal us about spasms and heart attacks! It is also not by chance that aortic stenosis, problems with the aortic valve, and its fibrosis occur. All these are links in one chain: a periodically occurring lack of arterial blood volume.

How can these diseases be prevented? It is necessary to reduce the impact of stress by somehow replenishing the arterial bed with blood. If the proposed theory is correct, then it becomes clear why all breathing exercises, including special oriental breathing practices, are so popular for maintaining health. During breathing exercises that directly affect the lung tissue, i.e. on the pulmonary circulation, there is a more active replenishment of the volume of the arterial bed and the negative pressure in the large arteries and aorta is removed, health improves! Almost any physical exercise is also useful, because they do not exist without useful loads on the breathing apparatus.

If your volume of the arterial bed is completely filled with arterial blood, then there will be no negative stresses in the walls of the aorta and large arteries, which means that atherosclerosis will not arise or progress!

All arguments and reasoning in this article are indirectly confirmed by scientists who have dedicated their lives to understanding the nature of atherosclerosis.

Let's list some scientists.

1) A widely known concept of the development of atherosclerosis is the "Response to Damage" hypothesis, formulated by the American researcher R. Ross [5] in 1976.

2) In 2003, academician E.I. Chazov published data that proved that neither obesity, nor smoking, nor increased cholesterol are directly related to the increase in mortality from CVD and that the cause of CVD should be sought in the consequences of stress [6].

3) Atherosclerosis is primarily an inflammatory disease as a response to damage to the arteries [7, 8]. Cholesterol plays a minor role. Atherosclerosis is a focal disease, not a diffuse one. If this were not so, then the walls of the arteries would simply absorb cholesterol along their entire length! But statistics show that plaques have preferred locations. The author of this article found answers to the following key questions. Where might there be preferential sites for plaque growth? What mechanism and what force causes fractions of cholesterol (LDL) to penetrate into the walls of arteries and cause the growth of plaques, what is the nature of this mysterious infiltration? What physical force leads to endothelial dysfunction? Having answered these questions, the nature of atherosclerosis and, as a consequence, many CVDs becomes clear.

Conclusions

In order to keep the cardiovascular system healthy, it is necessary, as far as possible, to avoid stressful psychological situations and in some way to escape from stress, avoiding loss of arterial blood. A healthy lifestyle, physical and breathing exercises are necessary, which increase blood flow through the pulmonary circulation and restore the balance of blood volumes in the arterial bed. It must be admitted that all breathing practices help replenish the volume of the arterial bed, so they are useful to one degree or another. All this is confirmed by practice and, most importantly, by the new theory of atherosclerosis presented here. The author of the article found the answer to the main question of cardiology of the 19th, 20th and 21st centuries: "What "force" causes cholesterol fragments from the main arterial flow to penetrate into the walls of the arteries?" Heart inventors and researchers need to focus their attention on two areas: working to monitor arterial blood leaks and maintaining the required arterial blood volume individually for each person. A wide field for creativity opens up here.

Total

I believe that about 200 years of continuous search by all countries for the causes of atherosclerosis have ended. The reason has become known since 2020 is the uncontrolled loss of arterial blood volume in humans during stressful influences! My confidence increased every year after 2020, because during these 4 years, none of the academicians of medicine expressed a single critical word to me.

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