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Review Article

# Proof of the cause of atherosclerosis, "standing on the shoulders of giants" Vladimir Ivanovich Ermoshkin

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**Abstract**

The article is devoted to the most important topic in medicine: the cause and mechanism of atherosclerosis. It is believed that the health of each person and their life expectancy depend on the degree of atherosclerosis.

**Keywords:** atherosclerosis; arteriovenous anastomoses; arterial blood volume; arterial spasm; arterial and aortic dissection; blood stagnation

## Objective

The cause of atherosclerosis is the main problem that modern medicine has been solving for many years. "Giants of medicine" have proposed and "proven" more than a dozen hypotheses, at least over the last two centuries, which have never been accepted as the main and only "correct" theory.

In this article, we try to describe and substantiate the main and only cause of atherosclerosis [1-10, 16, 17].

As for the prevention and treatment of atherosclerosis, this is a completely different task and this task still needs to be worked on, but the direction of the search is already known - monitoring the volume of arterial blood and maintaining the decreasing volume at a certain level calculated for a given person.

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

## Results.

Due to its powerlessness in the face of the problem and the inability to choose the main theory from the list of theories or combine all the proposed theories of atherosclerosis into one theory, modern medicine still admits that a person may have several causes of atherosclerosis, and lists a long list of them. Below is a somewhat shortened list of causes of atherosclerosis. There are three such theories, and they are the most common in medical literature. According to the author, the combined popularity of these three causes relative to other causes is more than 90%.

A) Psychoemotional theory - due to periodic stress.

B) Theory of endothelial dysfunction - primary disruption of the protective properties of the endothelium and its mediators.

C) Theory of lipoprotein infiltration - primary penetration and accumulation of lipoproteins in the vascular wall.

**Additional observations**. Based on the results of a long-term study of the causes of atherosclerosis, medicine has established that the primary "risk factors" of this disease are: age, smoking, alcohol, hyperlipoproteinemia, arterial hypertension, diabetes mellitus, obesity, physical inactivity, emotional stress, poor nutrition, heredity, post menopause, hyperfibrinogenemia, homocystinuria, hypothyroidism, irregular sleep.

The three "theories" listed above cannot be built into each other. It is necessary to prove and choose: either one theory is correct or the other, or to put forward a new theory, a universal one. And do not forget about the contradiction: "When there are many theories, it means that none are correct." Let us name some of the leading scientists, including Nobel laureates, who made the greatest contribution to the study of the causes and mechanisms of atherosclerosis: Jean-Frederic Lobstein, N.N. Anichkov, Rudolf Virchow, A.L. Myasnikov, E.I. Chazov, I.V. Davydovsky, G.F. Lang, Takio Shimamoto, M.S. Brown, J.L. Goldstein, R. Ross, J.B. Muhlestein, D. Fredrickson and others.

On the other hand, 13 Nobel Prizes were awarded for the study of cholesterol during the 20th century. The increased interest of scientists in this fat-like substance is clearly not accidental!

By the way, Alfred Nobel, who established the prize in medicine, himself suffered from some diseases. Alfred Nobel died on the night of December 10, 1896, at the age of 63. Judging by the clinical picture of the disease and its dynamics (a two-year history of stable angina with its progression and the development of ischemic stroke against this background with its characteristic clinical picture), Alfred Nobel's disease was based on ATHEROSCLEROSIS with damage to the coronary and cerebral vessels.

About 130 years have passed since then...

By 2020, unfortunately, it could be stated that the "giants of medicine" have not achieved significant real results in the search for the causes of atherosclerosis. In the end, at the beginning of the 21st century, medicine spontaneously, without being guided by formal logic, declares that such concepts as "polygenic disease" and "multifactorial disease" are synonyms, and the incidence of atherosclerosis depends on "gene defects". And these defects within different populations across continents are distributed according to different laws! As a result, the volume of research in the coming years for official medicine was planned to be enormous, if right now we do not suggest that it stop.

This is the result of the search by mid-2024. Meanwhile, the leadership of medicine does not react in any way to the new theory of atherosclerosis development, which arose 4 years ago in 2020 [1-10, 16, 17] due to blood leaks from the arterial pool. Let us note here that this article is devoted to the development of atherosclerosis in large arteries of the upper half of the body; in the lower half of the body, atherosclerosis develops later and with some distinctive features that are not considered in this article.

And now, in July 2024, “standing on the shoulders of the giants of medicine,” the author of this article, a lone physicist, must substantiate the main links for the synthesis of the correct theory of atherosclerosis, and introduce the missing arguments to prove the only cause of atherosclerosis. The new theory should not contradict any fact statistically observed by researchers, and should certainly include at least the three most popular “private theories” and take into account the numerous “risk factors” presented above.

*Hypothesis of the cause and mechanism of development*

So, the new theory of 2020 claims that the cause of atherosclerosis is the lack of arterial blood volume in the arterial bed in humans at some points in time, created by the cardiovascular system. Note that the total volume of circulating blood in all arteries, veins and depots at some point in time may be normal or even excessive, but the arterial blood volume is insufficient for several hours (or minutes) due to blood leaks through open arteriovenous anastomoses! Arteriovenous anastomoses, working as emergency valves, can periodically open and close due to fluctuations in blood pressure (BP) in the area of these "shunts". It can be assumed that each adult at some stage of life has their own optimal (on average) volume of the arterial bed. Loss of arterial blood due to leaks through anastomoses is caused by stressful situations with an increase in blood pressure. Stressful situations may include: nervous tension at home or at work, everyday worries about children and parents, conflicts, monotony and duration of the work itself, tension when speaking in front of an audience, other fears, excessive joy and other loads on the central nervous system. Such stress loads may periodically occur throughout life - this is a common situation for an ordinary person, but much depends on the nature of the nervous system of this person and his commitment to physical and breathing exercises, proper rest. The balance of arterial and venous blood is gradually restored with moderate physical and respiratory exercise and / or in a lying position during rest. Let's assume that arterial blood leaks, apparently, can often occur at work or at home in a sitting position in front of the TV or computer, i.e. in the absence of physical activity for several hours. Frequent leaks of arterial blood into the veins create an overflow of the venous pool, venous and lymphatic congestion, especially in the lower half of the body due to the effect of gravity. The horizontal position of the body during the night's sleep does not always rid the body of excess stagnant venous blood and intercellular fluid. Gradually, congestion in the pelvis and lower extremities can provoke lymph congestion, weight gain, obesity, and the development of diabetes. In such cases, a standard blood test shows an elevated level of stagnant glucose (HbA1C) and other abnormalities. The situation is somewhat alleviated by the procedure of Arabic hijama or hirudotherapy, but medicine does not recommend them, since such procedures can reduce the number of patients and the income of doctors.

***Proof of the cause of atherosclerosis***

The proof is the very life of most people: atherosclerosis develops in everyone! But some have more vascular damage, while others have less. Let's consider what happens when the volume of arterial blood decreases due to blood leaks. Since blood is incompressible, this leads to the same decrease in the volume of the arterial bed. At the same time, do not forget about Harvey's law. It should be taken into account that the current volume of the arterial bed is always equal to the current volume of arterial blood. If a person's arterial blood volume increases over several minutes or hours, the volume of the arterial bed also increases, and if the volume of arterial blood decreases, the volume of the arterial bed also decreases by the same amount! In young or middle age, soft arteries without signs of atherosclerosis, with some loss of arterial blood volume (for example, due to nervous or physical stress), temporarily reduce the cross-section of their lumens due to radial stretching of the muscular layer of the walls. Since the outer layer of the arteries, called "adventitia", is practically inextensible, the middle layer is subject to stretching and the expansion occurs in the direction of the center of the arteries. In other words, this is how physical "tearing forces" of the endothelium from the muscular layer of the arterial walls act. This is why dissection of the walls of large arteries and the aorta can occur!

In other words, a spasm occurs in the large arteries, but a spasm initiated by physical forces. During such a spasm, the muscular layer of the arteries increases in thickness, while the lumen of the vessel decreases. Under stress, these physical forces primarily reduce the lumens of the large arteries near and, especially, slightly above the myocardium. These vessels include the initial sections of the aorta, carotid, vertebral and coronary arteries. Why is this so? Because this is how gravity acts on vertically located arteries. (In animals, such a concentrated effect does not occur due to the horizontal position of the spine and all major arteries.) The forced reduction in the lumens of the large arteries leads to a decrease in the area of the endothelial lining, which in turn leads to a decrease in the distance between the flat cells of the endothelium, up to their shifts and detachment. With frequent and significant spasms, endothelial alteration occurs over the years. Gradually, the smooth inner layer of the arteries becomes rough due to constant damage, inflammation and healing of the arterial walls. These events lead to a constant increase in the level of C-reactive protein. When the described events, stress and forced spasms, occur frequently in the arterial system, then gradually, along with the years lived, "dysfunction of the endothelium" occurs, the arteries decrease their lumen every year, lose elasticity, and heart failure develops. The cardiac output of blood portions becomes increasingly limited, since due to the frequent lack of arterial blood, the aortic valve, aortic arch and large arteries become more rigid and resist the ejection of previous portions of blood. Each microtrauma of the endothelium turns into a scar. And the resulting scars, as we know, leave an imprint for life. But the main thing that can happen in the walls of the arteries is the formation of small, and then large plaques. Plaques are located in favorite places of arteries, where negative, relatively normal, local pressure impulses often occur due to the action of pulse waves. Maximum damage to large arteries and the aortic valve occurs, firstly, with arterial blood leaks and, secondly, with maximum compression of the arteries in diastole (but not in systole, as modern medicine believes: "due to high blood pressure"). The vessel reduces the lumen, and at the same time, negative pressure occurs in the expanding muscular layer of the arteries. With each diastole, negative tissue pressure occurs in the walls of large arteries when it is stretched, as

a result of which there is a "suction, in other words, suction" of blood from the main flow. Such processes in the walls of the arteries are similar to the work of a pulse vacuum cleaner! Due to negative pressure impulses, any light fractions of the blood penetrate through the "holes or cracks between the endothelial cells" into the muscular layer and are fixed there. It is precisely these forced penetrations of various blood particles, mainly lipoproteins, that medicine calls "infiltration"! First of all, low-density lipoproteins (LDL) penetrate under the endothelium into the muscular layer of the arteries. The lipoprotein particles themselves are not the cause of atherosclerosis, they are neutral in relation to the endothelium, they only respond to the pressure difference between the flow and the muscular layer of the walls. This is a forced movement. Other blood particles also react to the same pressure difference, but more weakly: triglycerides, phospholipids, proteins, etc. It turns out that the level of LDL, not being a direct cause of atherosclerosis, determines only the rate of plaque formation! Therefore, reducing the level of LDL is the right step in the fight against atherosclerosis in conditions when uncontrolled leaks of arterial blood often occur, i.e. during stressful moments.

Another fact. Growing plaques reduce the lumen of large arteries, and in the narrowed lumen of the arteries, the blood flow velocity increases and the external pressure relative to the inside of the plaque decreases. Because of this, additional physical forces arise to tear off the top of the plaque from the walls of the arteries. Because of these forces, a large plaque can ulcerate, and then a microthrombus can form. Such a thrombus can block any artery and cause a stroke or heart attack. So, the main thing: infiltration is created by physical suction forces, this is not a common metabolic disorder! This is how plaques are formed, and they usually grow, because stress in a person can continue, and, unfortunately, no one believes in effective methods of counteracting stress, since there is no correct and unified theory of atherosclerosis. Each doctor talks about his own version of the disease and treatment. People believe only in pills and operations such as installing stents - this is how big MEDICINE has taught them over many years. But such MEDICINE in the interpretation of the causality of some cardiovascular diseases remained, as we find out, erroneous!

With significant blood leaks through anastomoses, arterial pressure can increase somewhat, since it becomes more difficult to deliver arterial blood, the volume of which decreases, to the brain, the pressure in the vasomotor center drops and the vasomotor center reacts to this. With large blood leaks, so-called hypertensive crises can occur! Crises tell us that the general decrease in arterial blood has reached the limit of what is possible, arterioles and small arteries of secondary vessels are compressed to the limit (while the hands and feet are always cold). The remaining volume of arterial blood is barely enough to provide the human brain with minimal nutrition. At this time, sticky cold sweat is formed not from the arterial pool, but from overflowing pools with stagnant intercellular fluid and from the veins. During a crisis, the patient should be in a horizontal position to weaken the effect of gravity. It is necessary to give an injection or take medications to force the arterioles and small arteries to open, after which (presumably) venous blood and interstitial fluid will replenish the arterial bed in reverse. Blood circulation and organ function will be restored, hot blood will warm all organs again. A person's life will be saved.

In order to quickly replenish the volume of arterial blood and thereby slow down the development of atherosclerosis, it is necessary to increase the blood supply through the pulmonary circulation, through the lungs, to the left ventricle and aorta during times of stress. Now it becomes clear why most ancient teachings on health and simple breathing practices bring a positive effect: with the help of these practices, the volume of arterial blood is replenished, vascular spasms are relieved, and blood pressure is normalized! The new theory of atherosclerosis confirms this!

In conclusion, the author suggests conducting a physical natural modeling of the occurrence of atherosclerosis, on a sample of some small animals, such as guinea pigs, hamsters, Chilean degus, rabbits, miniature pigs or others. There is no need to feed animals cholesterol, which is essentially nonsense! Animals should be placed in a vertical position for 3-6 months, releasing them from confinement every day several times for several minutes to take their usual food.

I believe that we need to hurry with these works, no one will give modern doctors another 100 years of reflection, as is customary in medicine.

## Conclusions

Until 2020, all the work of the "great" researchers of the causes of atherosclerosis, i.e. the "giants of medicine", was essentially an attempt to search for and describe continuous medical, biological, chemical, genetic, age-related changes [11-15] in the cardiovascular and other human systems under the continuous destructive concentrated impact of mysterious physical forces. The presence of these forces was unknown to doctors and was not taken into account by cardiologists for many decades.

In the process of proving the new theory of atherosclerosis due to periodic losses of volume (EXACTLY THE CURRENT VOLUME!) of arterial blood, it was possible to logically substantiate that the new theory, as more universal, organically includes in its composition at least three of the most popular theories (already as factors): psychoemotional, endothelial dysfunction, lipoprotein infiltration [1-10, 16, 17]. So, we can conclude that the cause of atherosclerosis has been known since 2020, but the "giants of medicine" have not noticed it yet.

As for the prevention and "treatment" of atherosclerosis, there is a lot of work to be done in this area. The main thing is: constant monitoring of blood volume, replenishment of blood volume in the arterial bed plus removal of "excess" fluid from the venous and lymphatic beds, stress management, physical activity, breathing and other exercises. As a result: the development of atherosclerosis will be slowed down for several years, life expectancy will increase. This is the opinion of the author of the article. It took the author 12 years to study cardiology and to create and substantiate a new theory of atherosclerosis.

**Afterword.** When the article was already ready, a message came from Japan [18], which indirectly confirms the new theory. Researchers from Shinshu University have developed a DNA aptamer called iSN04, which affects the nucleolin protein in arterial smooth muscle cells and counteracts it, i.e. "spasm." In other words, iSN04 counteracts the "mysterious physical forces" that the new theory of atherosclerosis suggests. The antinucleolin aptamer helps maintain smooth muscle cells in a differentiated state, offering new potential for treating atherosclerosis and other vascular diseases. But experimental data from Shinshu University are needed to draw a final conclusion. It is necessary to wait and, most importantly, find an answer to the question: if iSN04 acts on all arteries simultaneously, what will happen to the patient's other organs where there were no plaques?

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