ISSN: 3049-5849

Journal of Vascular Medicine & Surgeries

SCIENTIFIC Research and Community

Research Article

Open Access

Why do Atherosclerotic Plaques Form in the Arteries, but not in the Veins?

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Received: January 05, 2026; **Accepted:** February 10, 2026; **Published:** February 20, 2026

Introduction

Blood flows in the arteries after it is saturated with nutrients in the intestinal walls and oxygen in the lungs. Arterial blood is scarlet, giving energy and life to every cell of the body. The blood in the veins is already spent, poor in nutrients, saturated with carbon dioxide, and there is little oxygen in it. But for some reason that is still unknown to medicine, atherosclerosis and plaques form in the arteries, but there are no plaques in the veins. Why is this happening? Researchers have been thinking about this question for at least the last 100 years, but so far there has been no result.

The Purpose of the Research

The aim of the research was to find the reason why plaques form in the arteries, but there are no signs of atherosclerosis and plaques in the veins.

Methods

An attempt is made to find physical differences in the functioning of arterial and venous channels. It was necessary to evaluate in various situations the thickness and elasticity of the walls, the speed of blood movement, the possibility of changing vessel profiles, and the throughput of different vessels. It was necessary to find differences in the work of the walls of arteries and veins in the presence of blood flow in them.

Results

This work is another attempt at theoretical and practical research of human blood vessels. The article also addresses the issue of the cause and mechanism of atherosclerosis! Since this research paper continuously compares geometric, mechanical and hemodynamic differences between arterial and venous basins as the material is presented, it is inappropriate to divide the text into two parts: a section on arteries and a section on veins, in the author's opinion.

The arterial pool in a healthy person has a very small volume of 850-950 ml, and the venous one is about 4,500 ml or higher, i.e. about five times larger. This means that the entire volume of arterial blood in humans is completely renewed 5 times faster than the volume of venous blood.

The largest human artery, the aorta, reaches a diameter of about 3 cm. The largest vena cava reaches a diameter of about 3-3.5 cm at its confluence with the right atrium.

All other vessels have significantly smaller transverse dimensions. The blood flow rate in the aorta is 20-25 cm/sec, the linear velocity in the arteries is 30-50 cm/sec, the velocity in the capillaries is 0.03-0.05 cm/sec, in the vena cava 10-25 cm/sec, and in the small and medium veins 1-14 cm/sec.

The speed of blood movement is influenced by: the cross-sectional area of the vessel, the resistance of the vascular walls, and the viscosity of the blood. It was possible to find special differences in the work of these vessels. The main difference is that arteries and veins provide blood vessels with blood and regulate blood flow in different ways.

Let's look at what happens in the arteries and veins from the point of view of physics, or its special section "hydraulics". What is the difference between the venous blood pool and the arterial pool? The pressure in the veins is low and is equal to 8-20 mmHg.

The total filling of the venous bed is always less than 100% possible or more than 100%, the venous is elastic and can stretch. If you do not monitor the health of the veins, varicose veins, valve diseases and other vein problems may occur.

So, what happens when there is an additional influx or outflow of blood into the veins? With additional blood flow, the vein section simply takes on a more rounded cross-sectional shape with the maximum capacity of the venous bed. But with a lack of blood in any part of any vein, the latter changes its profile and thereby adjusts to the reduced volume! A particular vein is "squeezed" from two opposite sides and turns into a vein with an "oval profile".

The veins adjust to the required throughput based on the current volume of venous blood in a particular area. Veins reduce the cross-section of the vessel and the capacity of the vessel by changing the transverse profile of the vessel, for example, from a circle to an oval. And vice versa: veins can increase the capacity of the vessel by changing the profile from oval to circle.

Note that when the envelopes of the same vessel are equal, a circle always has a larger cross-sectional area and carrying capacity than an oval!

Arterial pressure in the aorta and in the large arteries near the aortic valve is significantly higher and approximately equal to 120/80 mmHg. At the end of the arterial flow, the residual pressure is about or less than 15-20 mmHg.

Healthy elastic arteries always maintain a rounded section of the vessel, even with a slight decrease in blood volume, only slightly reducing the inner diameter by stretching the muscle layer of the arteries inside. The external diameter of the artery changes very little.

All arterial blood is limited by elastic three-layered arterial walls consisting of adventitia, muscle layer, intima. The outermost inner layer consists of a thin and smooth unicellular endothelium.

The venous system is also limited by three-layered walls. But all the layers of the vein walls are thinner. For example, the muscle layer in the veins is 0.5 mm, and in the arteries 1.0 mm.
The arterial basin (from the aortic valve to all arterioles) is almost constant in volume. With a significant increase in blood pressure (BP), one of the arteriovenous anastomoses can open and discharge some of the arterial blood, say 5-10%, into the venous basin.

For example, an anastomosis is known between the superior mesenteric artery and the portal vein [1, 2]. After opening the anastomosis opening, the pressure in the arterial system decreases, then the opening closes. It can be assumed that such an anastomosis works as an emergency valve.

The venous basin (from all venules to the tricuspid heart valve) has the ability to vary its volume over a wide range (for example, it can increase the volume from 4,500 ml to 5,000-6,000 ml, i.e. up to 30%). At the same time, we do not forget about the permanent law of Harvey - the law on equal capacity through the left and right ventricles.

A person does not experience any particular inconvenience with a decrease or increase in venous blood volume. Although venous congestion and swelling may occur periodically, more often in the lower half of the body.

What happens during blood loss in the arterial bed? Recall that the transverse contour of the walls of any artery is close to the circumference [3].

As a result of the loss of blood volume, there is a negative (relative to the current average) pressure in the arteries.

Then there is a forced spasm of elastic arteries. The walls of the arteries stretch laterally, towards the center of the artery. Basically, the middle muscle layer stretches. The stretching is directed inward, towards the center of the artery. The magnitude of the spasm is proportional to the volume of arterial blood loss. First of all, spasm occurs in the aorta and in large arteries near or above the level of the heart, where the speed and volume of blood movement are maximum. Limb arterioles spasm requires a longer period of stress and lack of physical activity. Partial relief of all types of arterial spasm are a well-known breathing exercise.

So, spasms are caused by physical forces acting on the inner wall of the arteries towards the center of the arteries, while the lumen of the arteries decreases slightly, and the thickness of the walls increases due to their stretching. The “forces of separation” of the endothelium from the intima act [4, 5]. All this contributes to damage to the inner layer of the arteries, including alterations and endothelial dysfunction. In parallel, stretching of the muscle layer and other layers from the main stream produces absorption of the lightest blood fractions, mainly LDL, into the muscular layer of the arteries through holes and crevices of the endothelium. The “suction forces” are equal to the “separation forces” and these forces have opposite signs!

Veins, unlike arteries, reduce the vessel’s capacity by changing the transverse profile of the vessel, for example, from round to oval [6, 7]. As a result, there are no forces of separation of the endothelium from the middle layer in the walls of the veins, the endothelium is not damaged, there is no reason for atherosclerosis in the veins . That’s why there are no plaques in the veins!

Due to frequent leaks of arterial blood into the venous basin, the structure of the wall layers is damaged and inflamed. Of course, arterial damage and plaque growth occur over a long period of time, over months and years. The result: a constant increase in C-reactive protein, and over time, serious areas of atherosclerosis and plaque growth. Most often, plaques are located precisely at the points of maximum pressure drop along the flow, i.e. at the points of maximum arterial spasm known to doctors due to arterial blood leaks. These sections of the arteries are characterized by low hydraulic strength of the vessel, these are points of bifurcation, or points of changes in the diameters of the arteries and bends of the current.

Why do plaques sometimes rupture? Physics gives the answer, but more precisely, hydraulics again. If the plaque protrudes into the lumen of the artery, the area of the internal lumen of the vessel usually decreases in this place. According to hydraulic formulas, the pressure decreases above the top of the plaque, and the pressure increases before and after the plaque. This means that as any plaque grows, the blood flow gradually increases the forces of plaque detachment! That’s why calcification of the plaque top is so important!

Now there is a major medical issue that needs to be resolved. How to resist the onset of atherosclerosis? The answer, apparently, is this: it is necessary to quickly replenish the arterial blood volume in some way after blood leaks. How to do it? I believe that there are still many medical problems to be solved here: to create a leak monitoring system and a system for replenishing the volume of arterial blood to normal. What will it do? This is expected to delay the rate of plaque growth and increase human life expectancy.

The results of this research paper confirm a popular observation: “All diseases are caused by nerves”! Stress and stress lead to an increase in blood pressure, leakage of arterial blood volume and arterial spasms. Arterial spasm, especially at the bifurcation points, leads to rapid plaque growth. Plaque growth leads to organ ischemia, various diseases of the cardiovascular system, heart attacks, strokes, and shortened life expectancy.

Conclusions

- In the 5 years since 2020, it can be considered proven that under certain conditions, plaques begin to grow in the arterial bed, especially at the bifurcation points. The reason: leakage of arterial blood through anastomoses. Further, when the arteries spasm, impulses of suction force occur in the expanding muscular layer of the artery wall, which draw the lightest fractions of blood from the main stream. These include LDL and some other fractions most often.
- It can also be considered proven that similar plaques and atherosclerosis should not occur in the venous bed. There are

no spasms in the veins under normal conditions. And there are no suction forces from the flow into the vein wall. There is no physical reason for plaque growth.

- According to the New Theory, it becomes clear that in order to increase life expectancy, it is necessary to conduct research on monitoring one's own arterial blood volume and its "rapid" replenishment, for example, through breathing exercises or other as yet unknown methods!
- The author believes that in 2026, medical science will come to a revision of the causes and mechanism of atherosclerosis. The time has already come!

References

1. Rudenko M Yu, Zernov V A, Ermoshkin V I (2020) Cardiometry. Fundamentals of theory and practice. Under the general editorship of M. Y. Rudenko. Russian.
2. (2009) Online resource. A clinical case. Russian https://valsalva.ru/viewtopic.php?t=1101&sid=137874936ec435e6be6626bf749f6a0f.
3. Ermoshkin Vladimir Ivanovich (2020) The mechanism of development of atherosclerosis and pathological spasms of human arteries. European Commission on Cardiology 7.9: 69-74.
4. Ermoshkin VI (2023) The main cause of most cardiovascular diseases and cancer is arterial blood flow into the veins. Research Gate.net https://www.researchgate.net/publication/375332632_The_main_cause_of_most_cardiovascular_diseases_and_cancer_is_leakage_of_arterial_blood_into_the_veins.
5. Vladimir Ivanovich Ermoshkin (2024) The proof of the causes of atherosclerosis, standing on the shoulders of giants. Scientific and Technical Conference Biomed J 58: 50502-50506.
6. Ermoshkin, Vladimir Ivanovich (2025) A refined mechanism of atherosclerosis development based on several previously proven theories, Research proposals. Ros NOU http://www.medlinks.ru/article.php?sid=113593&query=%E5%F0%EC%EE%F8%EA%E8%ED.
7. Ermoshkin VI (2024) Once again about the main cause of atherosclerosis. Cardiological Vascular Center 3: 1-3.