

The Mechanism of Atherosclerosis and Pathological Spasms of Human Arteries

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Abstract

Purpose: 9 years have passed since the New Theory of CVD began to be created. Published in English and Russian about 40 articles on this topic. The reaction of the leaders of Russian medicine has so far been zero. The New Theory develops on its own. The author of the Theory believes that it is time to make some generalizations and discuss the cause of the as yet unknown but deadly mechanism of “causeless” spasms of human arteries, including atherosclerosis of the arteries of the brain and coronary arteries.

Method: Study of numerous sources of information posted on the Internet. Discussion of proposed ideas at conferences, publication of original articles in Russian and English-language medical journals. To study the phenomenon of “blood pressure surges” we used the “Cardiocode” device, a Russian computer hemodynamic analyzer.

Results: Cardiovascular diseases are still the leading cause of death in humans. The main reason is atherosclerosis and associated complications (strokes, heart attacks). Atherothrombosis is often the cause of strokes and heart attacks. It is believed that in half of cases of heart or brain ischemia is not associated with the formation of a thrombus, it is associated with a lack of local blood circulation. This article discusses the causes and the main mechanism of local blood circulation disorders in the arterial bed.

First, we describe the point of view of official medicine on “causeless” arterial spasms.

Keywords: *Atherosclerosis; Spasms; Cardiovascular Diseases*

Vascular spasm is a pathological contraction of the muscle fibers of the arterial wall, which causes narrowing of the lumen and disruption of normal blood flow. It is believed that the basis of the spasm of the cerebral vessels, like the spasm of the arteries of other organs, is a violation of neurohumoral regulation. During an attack, the blood flow slows down, the brain or any other organ experiences oxygen deprivation. As a result, the characteristic symptomatology of organ ischemia appears, which for the brain in a certain percentage of cases can be irreversible, a stroke occurs, and for the coronary vessels, a heart attack occurs.

It is believed that the primary defect underlying many CVDs is vascular endothelial cell dysfunction.

The vascular endothelium is a hormonally active tissue. The unique position of endothelial cells at the border between circulating blood and tissues makes them the most vulnerable to various pathogenic factors in the systemic and tissue circulation. These cells are the first to meet with reactive free radicals, with oxidized low density lipoproteins, with hypercholesterolemia, with high hydrostatic pressure inside the vessels lining them (with arterial hypertension), with hyperglycemia (with diabetes mellitus). All these factors lead to damage to the vascular endothelium, to dysfunction of the endothelium, as an endocrine organ, to the accelerated development of angiopathies and atherosclerosis.

Antioxidants, which eliminate the damaging effects of oxidative stress on vascular cells, as well as drugs that increase the production of endogenous nitric oxide (NO), such as L-arginine, remain promising drugs that improve endothelial function.

The first reports of the discovery of the positive effects of nitric oxide on the walls of arteries appeared in the early 80s of the last century. The avalanche increase in the number of publications on the study of the role of nitric oxide in biological objects gave rise to the American Association for the Advancement of Science and the authoritative scientific journal "Science" to name nitric oxide (NO) by molecule of the year in 1992. It seemed that a little more time and the problem of atherosclerosis and atherothrombosis would be solved, but this did not happen.

The merit of the Nobel laureates F. Murad, R. Furchgott and L. Ignarro (1998) was that their fundamental research provided such a unification, which led to the formation of a new field - the biology of nitric oxide. Currently, research in this direction is still ongoing. There is a search for drugs that can normalize and balance endothelial functions.

So, to date, after the discovery of the effect on the arteries of the "magic molecule" of nitric oxide, about 40 years have passed. Despite significant advances in the treatment of certain diseases, current research shows that the incidence of atherosclerosis has not decreased. Still, to one degree or another, most people, starting from middle age, are faced with atherosclerosis. It can be stated that almost the entire adult population needs prevention and treatment.

In our time, there is an increase in the manifestation of micro-strokes and strokes due to atherosclerosis, even among young people, among students. Therefore, even young people need to prevent atherosclerosis. Atherosclerosis is a soil for many age-related problems: for strokes, heart attacks, heart ischemia, hypertension, memory loss, visual impairment, premature aging, etc. But most importantly, atherosclerosis is the cause of more than 50% of natural deaths in the adult population. This is "disease" number 1 of humanity.

In parallel, new methods of dealing with arterial spasms have appeared. For example, treatment by stenting the carotid or coronary arteries. It turned out that the installation of stents has both its advantages and most importantly, its disadvantages, for example, repeated vasospasm near the stent. This is a big disadvantage of using stents.

In addition, there is an unexplained medical paradox [1,2]: the administration of acetylcholine, a precursor of nitric oxide (NO), in healthy endothelium, at the same time its normal function causes expansion of the coronary arteries. And with the development of atherosclerosis or in the presence of coronary risk factors, paradoxical vasoconstriction is observed. How to explain this endothelial dysfunction? Medicine doesn't know yet.

Maybe it's time to pay attention to the New Theory of CVD, where there are new ideas?

Let's move on to the description of the point of view of the New Theory of CVD [3-14] on "causeless" arterial spasms and an increase in their rigidity.

Strictly speaking, this is still a hypothesis. It is believed that enormous financial resources of mankind have been spent on an unsuccessful search for a cure for spasms, but so far there has been little success.

This is the essence of the idea of the New CVD Theory. Many human health problems begin with periodic increases in blood pressure due to severe physical or psychological stress and/or physical inactivity, which sometimes leads to forced openings of arteriovenous anastomoses (AVA) in the human vascular system. AVAs usually open for a short time, it is at these moments that too high blood pressure drops, then, after a few seconds, the AVAs close again. After that, blood pressure may rise again. This is the mechanism of "blood pressure jumps". When the AVA opens, blood from the high pressure pool quickly flows into the low pressure pool, i.e. into the veins. For example, from the superior mesenteric artery to the portal vein [3]. In the figure to Articles [4,5] present ECG graphs with jumps in blood pressure, which are obtained on the "Cardiocode" device when opening/closing large AVA.

At the first stage of its development, the New Theory of CVD showed that when the vena cava overflows with additional volumes of blood flowing through the AVA, it is possible single or multiple run of the heart pulse along the contour of the vessels with the return of a mechanical wave to the myocardium. This mechanical wave each time causes extraordinary excitation of cardiomyocytes. Very often, this is how pathological tachycardia [5] and some other types of arrhythmias occur.

What is the reason and meaning of the opening of the AVA gaps in a healthy person? The main reason is the presence of stress, anxiety, especially with a long time a person is in a motionless position, for example, a sitting or standing position. The very opening of the AVA lumens saves the arteries of healthy people from overload, from excessive stretching of the arterial walls, from premature strokes and heart attacks. In fact, AVA are mechanical valves that limit the increase in blood pressure above a certain value! But over time, many other problems arise, for example, excessive.

“Blood deposition” in veins and organs, in addition, tissue edema, visceral obesity, as well as filling the space between adjacent organs with fluid, like ascites, are possible.

So, persistent or intermittent leakage of arterial blood through open arteriovenous anastomoses (AVA) decreases arterial volume and increases venous volume. The increase in venous blood volume after damage to the venous valves leads primarily to edema and varicose veins of the legs, and then to other organs.

With a healthy heart, at the exit from the right and left ventricles, due to the Frank-Starling law, the corresponding blood emissions are maintained; under normal conditions, these fractions are equal. With open AVA, with the help of CNS body adjustments, losses are compensated and the excess blood is periodically returned from the veins to the artery by a slight increase in blood flow through the pulmonary circulation. The question arises whether this is the reason for shortness of breath [15] and other disorders in the respiratory system due to the fact that additional blood is thrown into the small circle for a long time. Blood can also be retained there due to a decrease in the ejection fraction of the left ventricle, therefore, the bronchi overflow, and suffocation occurs.

In response to the next blood leaks, the body, regardless of the installed stents, begins to adapt each time to insufficient, i.e. to a decrease in arterial blood volume over time and to an increase in venous blood volume. Spasms of many arteries are forced, but in new locations. Physics cannot be fooled.

Let me remind you that in young and healthy people, arterial blood makes up only about 15% of the total blood volume. This blood is located in elastic arteries, i.e. tubes like rubber.

A small digression. Let's mentally imagine a situation with a lack of blood in the vessels. In this case, the arterial vessels will remain in the form of tubes with non-falling walls and gaping holes, and in the soft veins, especially in the wide ones, the walls will close, there will be virtually no internal hole.

Suppose the total blood volume in a healthy person is 5 liters, there is only 0.75 liters in the arteries, the rest is in the veins, in the venous depots, including in the tissues. It is a small part of blood (0.75l) that has mechanical energy and is under a pressure of 120/80 Hg. The constancy of the volume of this small part of blood is very critical to leaks, to its percentage reduction. The arterial part of the blood, it and only it, carries oxygen and nutrients to all organs, to all cells. Venous blood has little hydraulic energy, it is much more passive. Veins only collect and direct all venous blood to the atria, but, unfortunately, not always optimally and not always to the full, too much excess blood accumulates in persons with heart failure in the lower body (during the day). At night, some of the excess fluids flow to the upper body and, therefore, in the morning there are problems with the outflow of venous blood from the head.

It is blood leaks through AVA that lead to a decrease in the volume of all arterial blood (say, for example, from 0.75 to 0.55l) and if the amount of blood decreases, then the internal working volume of all arteries, of the entire arterial basin, also decreases. First of all, the

inner diameter of the arteries decreases, mainly due to stretching of the elastic intima. All this leads to a disturbance in the circulation of microvascular vessels, endothelial dysfunction occurs precisely because of mechanical influences: due to stretching (loosening) of the intima in the transverse direction, due to a decrease in the inner diameter of the arteries, due to a decrease in the area of the endothelium of the arteries. A decrease in the area of the endothelium disrupts the function of nitric oxide. In this case, the outer layer of the arteries, namely the adventitia, keeps the inner two layers from a critical decrease in the diameter of the artery.

The capacity of the heart always adjusts to the volume of blood in the arteries. Large (normal) internal volume of the arterial bed is consistent with a large the volume of the ejection fraction of the heart (55 - 85%), a small volume of the arterial bed is consistent with a small volume of EF (30 - 44%). After all, if (mentally) make a complete ejection (about $0.1 * 0.85 = 0.085l$) with a small internal volume, then the arteries and small arteries will expand from 0.55l to 0.635l (an increase of 16%) and can rupture, because additional volume blood will stretch the walls of the arteries by a significant amount, by 16%. It is very likely that full ejection is possible only with the maximum volume of the arterial basin, i.e. with relaxed arteries and arterioles. In this case, the expansion of the volume of the arterial bed will be from 0.75 to 0.835l, an increase of only 11%, which is more acceptable. It becomes clear that with "large" losses of arterial blood through the anastomoses, the left ventricular ejection fraction can decrease by up to 30%. And these reducing the emission fractions, the New CVD Theory has a logical, from the point of view of physics, justification.

Let me give you an example from physics. If you fill the bottle with water (analogue of arterial blood), then quickly turn it over and immerse the neck of the bottle (analogue of AVA) at least a couple of cm into a bowl of water (venous pool), the water will not pour out of the bottle. In order for something to spill out, it is necessary (mentally) to squeeze the walls of the bottle. A bottle with elastic walls (rubber) when pressed will shrink and reduce its volume, part of the water will flow out. The difference from reality is as follows: we press on the walls of the bottle from the outside, and in the arteries and the aorta, the force to reduce the diameter of the vessels is applied from the inside. It is to the endothelium that the suction force is applied by means of a negative pressure wave in the arteries, it is this force that drags the entire structure to the axis of the vessel: endothelial cells, intima and adventitia.

At the initial stage, this can be observed in various people in the form of numerous spasms of small arteries and arterioles: hands and feet become cold. Arterial tubes cannot collapse, they have a good muscle layer, the arteries are elastic in any direction relative to their optimal diameter: both in tension and compression.

We would like to emphasize that at any moment of time, the internal volume of the arterial bed and the volume of arterial blood are always equal to each other. To expand the internal volume of the arterial bed to the optimal value, or in other words to expand the arterial vessels, it is necessary to increase the volume of arterial blood! There is no other way! This must be remembered.

If a person has healthy arteries and is in a calm state, then the pulse dilation/compression of the vessels occurs around its optimal value and the endothelial reaction to nitric oxide (NO) is normal, not paradoxical. If there is not enough arterial blood in the arterial bed due to leaks through the AVA, then the tension/compression does not occur in the optimal range. The greater the loss of blood in the arteries, the more paradoxical the arterial response increases. a significant decrease in the diameter of the arteries worsens the nutrition of the vessels of the vessels, the endothelial cells close together to the limit, the gaps between the cells decrease (it is possible that the cells "crawl" over each other and lose their functionality) and therefore, blocks the endothelial reaction. If the volume of arterial blood is not replenished for a long time, then the configuration of the reduced arteries "freezes" - the body adapts in order to preserve the energy of the media muscles of vessels, this can be done only through the formation of fibrous tissue, calcification of the arteries, in other words, atherosclerosis, plaques occur. Surprisingly, almost all of this is due to a "simple" decrease in arterial blood volume! The main secret of cardiology is revealed!

It can be assumed that this is the unknown cause of spasm of all arteries, spasm of the heart valves, the occurrence of myocarditis and myocardiofibrosis. An insufficient volume of arterial blood cannot but lead to spasm of the arteries and a decrease in the volume of the

chamber of the left ventricle of the heart, and then the chamber of the right ventricle.

For many decades, doctors have been looking for the causes of spasms, atherosclerosis, plaque, occlusion and other narrowing in the arteries. They called this phenomenon asymptomatic, causeless. But the New CVD Theory easily reveals these reasons.

So, what is the way out of this vicious circle in case of vasospasm and organ ischemia? Where to begin?

First, it is necessary to prove that the described mechanism is confirmed. This can be shown in animals with dosed withdrawal of arterial blood and the movement of these volumes into the venous bed.

Possible prevention of atherosclerosis: Arterial blood loss should be monitored.

It is necessary to learn how to transfer the lost blood volumes from the veins into the arteries of the systemic circulation, possibly by direct transfusion.

It is necessary to learn how to manage AVA-anastomoses, or to block their work using artificial AVAs. Whether it will be in the mode of regular medical procedures or in the form of an automatic device is still unknown. This is a matter for the near future. This is the business of inventors and designers of medical devices.

At this stage, only prevention is possible, which follows from the New Theory of CVD. You need proper nutrition, regular exercise (especially light jogging for your pleasure), preferably periodic bloodletting of venous blood (or donation), vacuum cupping, leeches, Arab hijama, but with modern modifications. Breathing practices with holding the breath while exhaling are also helpful to increase blood CO₂ and dilate arteries. As before, apparently, ancient practices, such as yoga, qigong, and others, are useful, but this is almost tantamount to changing the way of life.

Conclusion

1. The article substantiates a new mechanism of pathological spasm of large arteries. It is as follows. Spasms occur when the volume of arterial blood decreases due to its overflow into the venous pool through the AVA anastomoses. For this reason, the internal volume of the entire arterial basin also decreases. A decrease in the arterial basin is the cause of spasms, narrowing in the arteries, because the arterial pool, due to the elasticity of the walls, is always consistent with the blood volume, because blood plasma is practically incompressible.
2. The author believes that the main medical mystery has been solved, the mystery of the causes of spasm of the arteries of the brain and ischemic strokes, many other diseases of the heart and the entire cardiovascular system. It has been shown that "innocent" and "invisible" blood leakage through the AVA anastomoses is a very dangerous phenomenon. Such leaks, according to the New Theory of CVD, lead to spasms of many arteries, overflow of the venous basin, strokes and heart attacks, to excess weight, to atherosclerosis, organ ischemia, heart failure, arrhythmias and many other diseases, including some types of cancer.
3. The author expects that the information provided in the article will generate great debate in the medical community.

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