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Opinion

Why Do Atherosclerotic Plaques Form in Arteries, But Not in Veins?

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How to reasonably answer the question in the title? Doctors, unfortunately, do not know the correct answer. They also do not know the answer to the question: why dysfunction of venous shunts occurs quite often during coronary artery bypass grafting (CABG). It turns out that veins that are resistant to the formation of plaques, when their segments are transplanted as shunts into the arterial bed, suddenly dramatically change their properties. Why does this happen? Many doctors ask this question.

But shunts made from sections of arteries behave much better and can protect people with angina and heart failure from heart attacks and other cardiovascular diseases (CVD) for a long time.

Let us remember that CABG is now recommended for millions of elderly people.

Problems with the functioning of venous shunts compared to arterial shunts do exist.

This is the truth that emerges about the work of CABG.

It turns out that "the pathophysiology of atrial fibrillation (AF) after CABG is not completely clear and has not been studied [1]." The general medical opinion regarding the causation of AF is very vague: structural changes in the atrial myocardium, history of arterial hypertension, fibrosis, myolysis, slowing of interatrial conduction velocity, dispersion of refractoriness, fragmented atrial activity, atrial ischemia, trauma, acute atrial stretch, metabolic and electrolyte disturbances, systemic inflammatory syndrome, reperfusion syndrome, hyperadrenergic status, etc. It turns out that there are a lot of reasons for postoperative AF, and the list presented can even be continued if desired.

"Postoperative AF most often occurs between 2 and 4 days after surgery, with a peak incidence on the second postoperative day. In 70% of patients, this complication develops before the end of the fourth postoperative day and in 94% of patients - before the end of the sixth postoperative day" [1]. "An analysis of domestic and world literature indicates a 30-60% risk of atrial fibrillation in adult patients who have undergone open heart surgery, even in the absence of arrhythmias initially (!) [2]."

"During the first year after coronary artery bypass grafting (CABG), up to 15% of vein grafts are occluded [3]. Subsequently, in the period from 1 to 6 years, the rate of graft occlusion is from 1 to 2% per year, and in the period from 6 to 10 years - 4% per year" [4]. "The consequence of shunt dysfunction for patients is the return of exertional angina, and repeated interventions are associated with a high risk of complications" [5]. Three discrete processes underlie vein graft failure: thrombosis, intimal hyperplasia, and atherosclerosis.

But before we begin to express a hypothesis about the paradox with plaques on venous shunts, a small author's digression is necessary. I repeat, everything written in this article below is a hypothesis. But this hypothesis brilliantly confirms the new theory of atherosclerosis [6, 7], according to which atherosclerosis and plaques arise due to a lack of arterial blood in the arterial bed. And the lack of arterial blood is due to physical and psychological stress with little daily physical activity!

So, how does the arterial blood pool differ from the venous pool? We are considering an adult of average height and average weight. The text of the article is divided into paragraphs and these paragraphs are numbered.

1) The arterial pool in a healthy person under normal conditions has a very small volume of 850-950 ml, and the venous pool - about 4500 ml and above, i.e. five times more.

2) Pressure in the aorta and in large arteries near the aortic valve is 120/80 mm Hg. At the end of the arterial flow, the residual pressure in the veins is about or less than 15 mm Hg. Blood pressure (BP) acts like a spring in a wind-up children's toy: until the spring relaxes, the toy has energy and works. It's the same with humans. The only "spring" is the

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elastic arterial system, constantly replenished with blood using the heart muscles.

3) All arterial blood is limited by elastic three-layer arterial walls, consisting of adventitia, muscle layer, endothelium, and venous blood is also limited by three-layer walls. But all layers of the vein walls are thinner, for example, the muscle layer in veins is 0.5 mm, in arteries 1.0 mm. At the same time, it should be clear that both pools must exactly coincide in volume with the volume of one's own blood - there is no "empty" space anywhere in the vessels.

4) The arterial basin (from the aortic valve to all arterioles) is almost constant in volume. If it decreases (on average) in moments of stress due to blood leaks through anastomoses into the veins, then, apparently, only by a small amount. It can be assumed that a decrease is possible only by 5-10% (which is equivalent to the total stretching of the layers of all arterial walls by the same 5-10%). The author did not find any evaluation data from other researchers. Apparently, slow blood loss may be accompanied by some increase in blood pressure. Loss of blood volume in the arteries above "a certain norm" can provoke hypertensive crises [6]. (Preceding sign: cold hands and feet.) All this is because with a smaller volume of arterial blood it is more and more difficult to provide the necessary nutrition to the brain and, most importantly, the vasomotor center!

5) The venous pool (from all venules to the tricuspid heart valve) has the ability to increase or decrease over a wide range (for example, it can increase from 4500 ml to 5000-6000 ml, i.e. by 33%). (Don't forget about Harvey's law). At the same time, a person does not experience any particular inconvenience with a decrease or increase in the volume of venous blood. Although "harmless" venous congestion and edema may periodically occur, more often in the lower half of the body.

6) What happens during stress and blood loss in the arterial bed? Let us recall that the transverse contour of the walls of any artery is close to a circle. It is known that blood in moments of stress and rising blood pressure can flow into the veins through arteriovenous anastomoses, losing its volume. As a result of loss of blood volume, negative (relative to average) pressure occurs in the arteries. Result: a forced spasm of the elastic arteries occurs. The walls of the arteries are stretched in the transverse direction. The middle muscle layer is mainly stretched. The stretch is directed inside 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 blood flow rates are maximum. (Spasm of small arteries and arterioles requires a longer period of stress. Relieving all types of spasms: 3-5 minutes of special breathing exercises and additional blood volume will flow from the pulmonary circle through the left ventricle into the systemic circulation). So, spasms are caused by physical forces acting on the walls of the arteries towards the center of the arteries, while the lumen of the arteries decreases somewhat, and the thickness of the walls increases due to stretching. "Tearing forces" of the endothelium from the muscle layer act. All this contributes to damage to the inner layer of the arteries, including alteration and endothelial dysfunction. In parallel, from the main flow, as a reaction to spasm, small and light fractions of blood, mainly LDL, are absorbed and retained in the muscle layer. The "suction forces" are equal to the "separation forces", but these forces have opposite signs! Due to tearing forces, the structure of the wall layers is damaged and inflamed. The result: a constant increase in C-reactive protein, and over time, atherosclerosis and plaque growth. Most often, plaques are located precisely in places of maximum pressure drop along the flow, i.e. at the points of maximum spasm of the arteries near the heart "known to doctors." As a rule, atherosclerosis of the arteries in the lower half of the human body, if it occurs, occurs years later, and the mechanism is different: not due to a lack of blood, but due to its stagnation.

7) What happens when there is a lack or excess of blood in any vein? When there is excess blood, the vein simply takes on a rounded shape with the maximum capacity of the venous bed. But if there is a lack of blood in any vein, the latter changes its profile and thereby adapts to the reduced volume! A specific vein is, as it were, "compressed" from two opposite sides and turns into a vein with an "oval profile". In this case, the cross-sectional circumference of the vein does not change, but the cross-sectional area and blood volume decrease! As a result, no forces arise to separate the endothelium from the middle layer in the walls of the veins, the endothelium is not damaged, and there is no reason for atherosclerosis in the veins.

8) So, the walls of the vein with a lack of blood do not experience any excess stress. This is how nature ordered it. But the walls of the artery are damaged due to loss of arterial blood: the growth of atherosclerosis and plaques! And all this is due to the rigid rounded shape of the adventitia, which maintains the necessary lumen and volume of the arterial bed given by nature! During the evolution of upright man, nature did not find another solution to this problem: and this solution should be considered optimal!

9) And now the main point. Why in practice are venous bypasses for CABG not as durable as arterial bypasses? Because the walls of the veins are not adapted to negative pressure in general and, especially, to powerful negative diastolic pressure in the arterial bed. As we know, the walls of a venous shunt solve this problem of blood deficiency by changing the profile. On the other hand, when there is negative (relative to average) pressure in the arterial bed, the walls of the venous shunt cannot hold any sufficient volume of blood, the walls of the shunt easily begin to "collapse" in the transverse direction, turning into an elongated oval in shape, critically reducing the lumen and causing angina attacks. In addition, because the walls of the shunt cannot hold any minimum required volume of arterial blood, then the shunt, due to the heart pulse running through the arteries and through the shunt that is not completely filled with blood, begins to generate additional mechanical fluctuations. This is why atrial fibrillation and other heart rhythm disturbances occur. As a result, the venous shunt is often damaged due to large arbitrary mechanical stresses; over time, intimal hyperplasia of the shunt occurs, "atherosclerosis" of the venous shunt occurs, thrombosis and complete closure of the shunt lumen are possible. This is an unfavorable development of the patient's postoperative condition.

Favorable development of the postoperative condition apparently occurs when the connective tissue scar of the blood vessels becomes coarser. "This may not occur until 4-6 months after CABG. During this period, young connective tissue grows, which, in the process of maturation, gradually deforms the surrounding tissues with a tightening fibrous scar, which can be observed from a two-week period. Later, the connective tissue scar becomes rougher [8]." This scar, in fact, can be a

good mechanical barrier that prevents the "collapse" of the shunt, and these scars also reflect and weaken the randomly generated mechanical waves along the shunt and therefore the initiation of fibrillations does not occur. This is exactly what is confirmed by statistics. Apparently, the patient only needs to survive the dangerous first 6 days [1,2], during which the primary fusion of the venous shunt sutures occurs!

Another hypothesis. It can be assumed that the formation of additional artificial incisions and scars (sutures) on the venous shunt will have a positive effect on the suppression of AF and on the survival of patients in the first 6 days after surgery. At least it has been proven that ablation of the left atrium has a positive effect.

Conclusion

Conclusions on this hypothesis should be made by specialists, doctors, and surgeons. The author is waiting for criticism. Millions of lives are at stake.

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