

Why Most Attempts at Heart Transplantation have Insurmountable Difficulties in the Postoperative Period

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Abstract

Earlier, in 2010-2011, our group of researchers proposed a New Theory of Cardiovascular Disease (CVD). According to this theory, many CVDs are caused by the malfunctioning of large arteriovenous anastomoses (AVA). Normally working AVA should open with a significant increase in blood pressure (BP) in order to ease pressure in the arteries and prevent damage to the walls of the arteries. But, on the other hand, AVA should be closed too on time, so that there are no significant losses of arterial blood and do not overload the venous pool.

Keywords: Heart transplantation; Arteriovenous anastomosis (AVA); Cardiomyopathy; Heart failure

Introduction

Apparently, a healthy person has anastomoses works "as a Swiss Watches", but in a person who leads an unhealthy lifestyle, AVA can be closed late, or be constantly open, even at the minimum value of its clearance. Blood leakage leads to many CVD [1-19]. It can be said that incorrectly working AVA [20] actually leads to deterioration in human health, and the deterioration can be rapid or slow, apparently, depending on the amount of blood leakage and the parallel increasing problems. It is shown, what specifically can arise with open AVA. These are arrhythmias of different kinds, violation of capillary blood circulation, other CVD.

The official cardiologist view on heart failure and cardiomyopathy

For example, edematous syndrome, as one of the leading manifestations of heart failure, attracted the attention of clinicians for a long time. You can recall the work of the outstanding physician Hippocrates. At the same time, the physiological basis for the development of edematous syndrome was developed in 1896 by EG Starling. He suggested that the formation of edema can be caused by an increase in hydrostatic pressure in both arterial and venous parts of the capillary [21]. Probably only because of open AVA anastomoses, because open AVA transmit high pressure to the veins, and then, over time, the pressure spreads not only to the right atrium, but also to the opposite direction, into small veins and venules, creating stasis in the capillaries.

So, modern cardiology believes that heart failure is a complex syndrome, associated with a decrease in the potential capacity of the heart for pumping blood. The disease is characterized, so-called, cardiomyopathy (CMP), the mechanism of which, unfortunately, is still unknown. The disease leads to violations of perfusion of tissues of various organs, to an increase in the chambers of the heart, the thickness of its walls, to edema, to physiological disturbances of filling or emptying the heart chambers and other problems. Thus, the key

problem of medicine - the causes and mechanism of the CMP - has not yet been resolved.

A few more words about the problem of the CMP [22,23]. The definition of "cardiomyopathy" is collective for a group of idiopathic (unknown origin) myocardial diseases, which are based on dystrophic and sclerotic processes in cardiac cells - cardiomyocytes. With CMP, the function of the ventricles of the heart always suffers. CMPs can be "primary" and "secondary". Causes of "secondary" CMP may be IHD, hypertension, atherosclerosis, vasculitis, myocarditis, tamponades, etc. CMP are divided into types: dilated (stagnant), hypertrophic, restrictive and arrhythmogenic. The etiology of the "primary" CMP has not been fully understood to date. Among the likely causes of the development of CMP are: viral infections, hereditary predisposition, myocarditis, cardiomyocyte damage by toxins and allergens, endocrine disruption, immune regulation.

Treatment of cardiomyopathies: Specific therapy of CMP is absent, therefore all medical measures are aimed at preventing incompatible with life complications. In exceptionally severe cases, surgical treatment of CMP: mitral valve prosthetics or heart transplantation. With respect to the prognosis, the course of CMP is extremely unfavorable: heart failure progresses steadily, the probability of arrhythmic, thromboembolic complications and sudden death is high. After the diagnosis of dilated CMP, the 5-year survival rate is 30%. With systematic treatment it is possible to stabilize the condition for an indefinite period. There are cases exceeding the 10-year survival of patients after cardiac transplantation.

Thus, in official medicine there is no good solution to the problem, there is a study of options for CMP, the conditions of occurrence, risk factors, work on the classification of the disease, careful description of the CMP clinic. But for the creation of new knowledge, in order for the cardiology to look like a science, a critical analysis and synthesis of new knowledge is necessary. Unfortunately, having prepared a descriptive part of the problem, the science "medicine" has not yet completed the last two steps.

A look at cardiomyopathy from the point of view of the New Theory of CVD

We emphasize that this article does not consider cases of congenital pathologies - this is a separate problem. So, from the point of view of the New Theory, the mechanism of heart failure is the following [1-19]. As a rule, in young people the pumping function of the heart is normal, but with lived years due to stress, due to physical or psychological overload, due to malnutrition and lifestyle, due to increased blood pressure, due to muscle de-training or overtraining, large and small arteriovenous anastomoses (AVA) can spontaneously open. The systemic arterial pressure drops, the stretching of the walls of the arteries sharply decreases, while the arterial blood flows into the veins, thus violating the optimal balance of arterial and venous blood volume (20% and 70%, which for the total, say, 6 liters of blood is 1200 ml and 4200 ml). Let's postulate that in a healthy person, throughput per unit of time (by during 1 minute) and for right and for left ventricles at the point at the exit from the ventricles are the same, i.e., there is postulate the continuity of the flow in both circles. If they are not equal, then it means that there is some transition period in the work of the cardiovascular system, and this period should "quickly" end.

Thus, the opening and closing AVA, as well as the gaping AVA, force the cardiovascular system to regulate the volume and throughput of the right and left ventricles. If this is not done, then the volume of arterial blood will decrease to a critical level and a lethal event may occur. To avoid imbalance, the right ventricle should increase the flow capacity, and the left one - either leave the former, or reduce the flow. This imbalance is regulated by the emission fractions, which varies with the leakage of blood through the AVA.

So, in the first stage of the disease, due to blood leakage through AVA in the vena cava, there is an increase in pressure at the entrance of the right atrium and additional work for the cardiac muscle. This is because it becomes necessary to increase the cardiac muscle and/or heart rate (heart rate) because of the additional stretching of cardiomyocytes (the Frank-Starling mechanism).

On the other hand, the general overload cannot fail to arise because the heart performs additional, prolonged, unfortunately, useless work on pumping blood through the AVA and a constant adjustment of the volumes of arterial and venous blood.

The situation is similar to that if a straight artificial channel with a large difference in height between the canal's beginning and its end appeared at the gyrus of the river, in the form of a large loop. Naturally, a part of the water, depending on the width and depth of the canal, would rush into the artificial channel, and the old bed (in the form of a loop) would considerably become shallow and eventually the water in it would become dirty.

It is the same in the circulatory system: many organs lying below the beginning of the "canal", in our case, below the point on the artery, where the AVA begins, and higher from the point of confluence into the veins would have been left without normal blood circulation, blood stasis, thrombi, necrosis cells and, as a result, infections.

So, to maintain the necessary systemic blood pressure, the heart is forced to do additional work, since part of the energy of the myocardium is wasted. In addition, in some venules of some organs (after damage to the venous valves), the pressure rises to a critical one, at which blockage of blood circulation occurs. In fact, the blockage is due to the decrease in pressure drop between arterioles and venules.

Due to the forces of gravity, primarily increased venous pressure and blood stasis occurs in the organs of the small pelvis and legs.

As mentioned above, there is an overflow of the hollow veins, increased pressure in the right atrial zone, problems with the heart valves. Results are violation of filling and emptying of chambers, pulmonary hypertension. In addition, because of the increase in the tone of the hollow veins, extraordinary heart excitements can occur from any part of the heart muscle, because of the passage of mechanical impulses that hit the heart. The mechanism of excitation of cardiomyocytes can alternate, i.e. be mixed: bioelectric from the sinus node, and mechanically induced from any part of the myocardium. This is the true mechanism of cardiac arrhythmia. There are no meaningful transitions controlling the rhythm of the heart to "bundles of His" and "Purkinje fibers".

According to the New Theory, the "ectopic focus" is a small area, a "point on the myocardium," where the concentration of energy of mechanical impulses is cyclically occurring. It is this effect that explains the equality of the QQ intervals on the ECG with paroxysmal tachycardia: a mechanical pulse wave runs through the same contour of blood vessels. Naturally, the point of concentration of mechanical energy can either stand still or drift, move along the myocardium without significant consequences (at the first stage of the disease) for the cardiomyocytes themselves, and for the conduction system, and for the sinus node. The arrhythmia attack disappears if the tone of the vessels and the degree of concentration of the mechanical impulses decrease to a value below a certain threshold, or the impulses move in time and begin to form in the refractory period.

Because of the blockage of blood circulation, edemas, venous thrombosis, renal failure, pulmonary insufficiency, lymphatic stasis, necrosis of some groups of cells may occur, which can trigger systemic inflammation of the body and cancer [7,11]. At the second stage, because of continuous overloads of the myocardium and coronary arteries, the problem of damage to the heart muscle is added to all problems, all signs of cardiomyopathy (CMP) develop. Because of slow or rapidly worsening indicators of the pump function of the heart, blood circulation in many organs becomes critically inadequate.

Apparently, from the second stage of deterioration of health, there are no methods of treating seriously damaged organs. A person can die not only because of purely cardiac causes, but, for example, because of kidney failure, systemic inflammation, a severed thrombus, which from the point of view of the New Theory seems to be logically justified. At the second stage of the disease development, heart transplantation is used to save a person's life. But the chances of living, for example, more than 5-10 years with a new heart are not great, because with abnormally working AVA, the new heart again works under conditions of overload due to useless blood pumping through AVA, plus concomitant diseases acquired earlier. Every year, the potential for health will deteriorate.

Thus, it is possible to draw a preliminary conclusion: "to detect" and "treat" should first of all anastomosis AVA. These are two very important tasks. It is open AVA lead to many problems related to human health. Apparently, open, or malfunctioning AVA, sooner or later can damage any initially healthy heart, bring it to a state characterized by the terms "heart failure" or "cardiomyopathy".

The problem of heart failure in most developed countries is exacerbated every year. We emphasize that at present the best method of treating cardiomyopathy in its acute form is heart transplantation. But there is a problem of immune and infectious rejection of any

foreign body, including the heart. Practice shows that this problem is solved with very great difficulties and not always successfully. It can be assumed that heart transplantation is not an optimal option to increase the life expectancy of all people, but only to increase the life of individuals.

Below I cited some facts confirming these pessimistic conclusions

Fact 1: *The eighth heart of David Rockefeller [24]. Why did the hundred and one billionaire die?*

American billionaire David Rockefeller died, at March 20, 2017, in his home in New York. He was 101 years old. This was reported by the New York Times. According to the representative of the Rockefeller family Fraser Seitel, chronic heart failure was the cause of death. It is also known that David Rockefeller has undergone two kidney transplantation operations.

Thus, David Rockefeller was transplanted by a young heart 7 times and the kidneys 2 times. On average, every 6 years, the billionaire received a new heart after his 61st birthday. But health problems continued to worsen. Each of the transplanted hearts, the life potential of which, apparently was no less than 10-20 years, quickly broke down. I suppose: because of incorrectly functioning or even yawning AVA, which were both the root cause, and the consequence of heart failure and the CMP of seven hearts plus the pathological effect of AVA on all other organs.

Fact 2: *From the magazine TIME on April 4, 1983[25]: "Last week the long battle ended. Clark, 62, died quietly of breathing problems, renal failure, inflammation of the large intestine, and low blood pressure." The official cause of death: "Vascular collapse due to functional failure of many organs. Undoubtedly, the experiment of Clarke will help doctors to design a better heart." "We learned more about Clarke over the past few months than we have done in the past 9 years with animals," says Harry Hastings of the Utah Medical Center, an artificial heart specialist.*

Thus, the artificial heart did not help cardinally. Yes, and could not help, the lack of perfusion of tissues because open anastomosis AVA, and not the heart itself! The heart itself can be the cause only as a consequence of open AVA, i.e., at the second stage or after the "primary" heart attack. As for the "reduced pressure", this is another confirmation of the New Theory: blood pressure is falling because of the long open or gaping AVA!

Fact 3: *In France, a third person died [26], who was implanted with an artificial heart. This is reported by the developer of the bio-prosthesis company Carmat. The death of a 74-year-old patient occurred on December 18, 2013. According to available information, the reason was the stop of breathing in the background of chronic kidney failure. The artificial heart was implanted patient on April 8. At the end of August, he returned home. Analyzes did not show that the functioning of the bio-prosthesis has anything to do with the patient's death. As noted, he suffered from a number of serious diseases, including kidney failure. "" The second man with an artificial heart died in France in early May. The 69-year-old man was taken to hospital due to circulatory failure. Doctors replaced him with an artificial heart, but the patient died. The first operation to install the heart of Carmat was performed on December 18, 2013. The 76-year-old man died 75 days after the operation."*

Again - insufficiency, but already renal, plus circulatory insufficiency, but all this can be a direct consequence of open AVAs!

Pessimistic conclusions of large modern scientists on the problem of heart transplantation

- "It would be better to set such a course, which would develop drugs that can prevent chronic diseases, such as cardiomyopathy (damage to the heart muscle) and cardiovascular diseases. If such work is not done, "writes Dr. Lewis Thomas, board member of the Memorial Sloan-Kettering Cancer Center," we will forever be stuck on this immensely expensive, ethically questionable half-hearted technologies".
- Dr. William Friedewald, deputy director of the National Heart, Lung and Blood Institute: "Of course, our goal is to prevent that in the future there will be no Barney Clark (see Fact 2), but at the moment it's only a dream."
- Professor of the First Moscow State Medical University named after I.M. Sechenov of the Ministry of Health of Russia Shevchenko Olga Pavlovna: "When in the future we finally understand the causes of heart failure and learn how to treat it, in general there will be no need for a heart transplant. But until then, we have to fight for the transplanted heart to work longer."

Conclusions of the New Theory

- Experiments with heart transplantation are a great success of modern cardiac surgery. The accumulated experience in cardio surgery now allows rendering assistance to almost hopeless patients. But the statistics on heart transplants and the findings of large scientists seem to indicate that the cause of heart failure and cardiomyopathy has not yet been found. This means that it is necessary to increase the life expectancy of people in a different way.
- In accordance the New Theory, replacing your own heart, either mechanically or transplanted from the donor, or made in the form of a "soft" bio-prosthesis, or grown from your own stem cells cannot solve the problem cardinally. The root cause of heart failure most often appears not to be in the heart itself, but in the abnormal work of the AVA anastomoses, leading to blockage of blood circulation. It is necessary to treat or replace these connecting vessels with artificial AVA, or find another solution. Then problems with the true insufficiency of the "heart" will be transferred to a few years later. For how many years specifically - is unknown to anyone.
- For many decades, official medicine says: the mechanism of almost all cardiovascular diseases is unknown, treatment is performed according to symptoms. Six years ago a hypothesis appeared, later called "The New Theory", dozens of publications in medical journals, there is great interest. But the leaders of "cardiology" do not see this, they do not need a New Theory. How can I explain this? After all, the unproven New Theory actually turned into an "open problem". According to the logic and scientific rules, the "open problem" should be solved, not ignored!

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