

One_2_Eng_Sergei_Cancer

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Letter to the Editor

I am a physicist from Russia and I have prepared a report for you, which can be the beginning of great changes not only in cardiology, but in medicine in general. Let's take a close look again at the problem of the causality of heart failure. Let us briefly see how the notions of the causes of HF developed over the past two centuries.

Since the time of Jean Nicolas Corvizar, and until recently, the blood circulation insufficiency has traditionally been defined as a pathological condition when the heart is unable to deliver to organs and tissues the blood quantity necessary for their normal function. The failure of the heart to ensure the proper level of blood circulation was constantly associated in the minds of doctors with a decrease in its propulsive (pumping) ability.

But it turned out that in about 1/3 of patients the cause of chronic CHF is a violation of the diastolic function of the myocardium rather than the systolic function, while its contractive ability remains unchanged. Therefore, to consider CHF as a manifestation of exclusively systolic dysfunction of the myocardium is fundamentally incorrect.

Later, it was shown that many clinical manifestations of CHF (edematous syndrome, palpitation, nycturia, etc.), its complications (sudden death due to arrhythmia paroxysm), and prognosis are determined not so much by hemodynamic dysfunctions as by pathological activation of neurohumoral regulatory systems.

Here is the definition belonging to the Milton Packer: "Congestive heart failure is a complex clinical syndrome when the left ventricular function and neurohumoral regulation of blood circulation are disturbed, accompanied by poor tolerance (physical) load, fluid retention and shortened life expectancy." All this is true, but why is it only a question of LV, and right ventricular failure? No answer.

The special literature often discusses the question: chronic HF and circulatory inefficiency - is it the same, or do these concepts differ in some ways in their content? Medicine consider: the same. The Russian cardiology center Bakulev believes that heart failure can be the main manifestation of almost all (!) heart diseases.

So, from modern clinical positions CHF represents a disease with a complex of characteristic symptoms (shortness of breath, fatigue, decreased physical activity, swelling, rapid heart rate, etc.), which are associated with inadequate perfusion of organs and tissues at rest or under load. Also, a characteristic sign of CHF is fluid retention in the body. It can be stated that there is no single, internationally recognized and accepted definition of CHF. In cardiology, it took more than 200 years to find an adequate definition, description of the disease, a list of causes and mechanisms of heart failure. There is no result yet.

So, let's move on to the most important thing. Question: how to link all the listed clinical observations into one logical explanation of the HF mechanism? Looking ahead, I would formulate a new definition. The HF and most of cardio-vascular disease (CVD) arise from too long and uncontrolled leakage of arterial blood into the venous pool due to a malfunction of the regulatory mechanisms for opening / closing large arteriovenous anastomoses (AVA) that leads to global health's problems.

I emphasize that the new theory is not yet universally recognized. I suggested that AVA is opened because of stress and large physical or psychological loads for a given person. The contribution is also made by hypertension, excessive unreasonable diet, inadequate daily exercise, alcohol abuse and some other factors. With an increase in arterial pressure (AP), due to the opening AVA, a part of the arterial blood is discharged into the venous pool. Thus, the arterial pressure drops for a few seconds, and the venous blood pressure rises. This reaction is like protecting the body from strokes and heart attacks. Usually with a healthy person the AVA are closed after a while. So, there are "jumps of arterial pressure". These phenomena were confirmed by rheograms obtained on the CARDIOCODE device in Russia.

What happens when the AVA are opened for a long time or when the AVA are gaping? First, there is a volumetric overflow of the venous pool. In addition, venous pressure increases in all organs, especially located below the point of confluence of AVA in the veins. The venous pressure increases in the right atrium, which can lead to a problem with the heart valves. The loads on the venous valves of all veins, especially the veins of the small pelvis and the veins of the legs, are critically increased. Pumping function of the heart can remain as before normal, but the heart in such cases, besides useful work, also performs a useless work - pumping blood through the AVA. Because of this, there are overloads of the myocardium and the ischemia of the heart is possible.

Periodically, from the overflowed vena cava to the right atrium, the mechanical waves generated by the pulse arterial wave from the AVA region and the liver begin to run up to the right atrium. These mechanical waves, when a certain threshold is exceeded by compression-stretching of cardiomyocytes (CMC), can begin the generation of mechanically induced excitations from any point of the myocardium, even from the apex of the heart. Sometimes on the ECG tape of a patient we can see a superposition of two excitations: both from the sinus node (narrow QRS), and from mechanical waves (usually wide QRS). Thus, the paroxysmal atrial and ventricular tachycardia attacks are generated, which can result in myocardial fibrillation and even sudden cardiac death (SCD).

Thus, in the non-optimal operation of AVA in humans, attacks of certain types of cardiac arrhythmia may occur due to mechanical effects on the myocardium. Medicine, while considering the causes of arrhythmia, has "forgotten" about mechanical stimulation. And this mistake is more than 100 years old!

At the same time, with prolonged AVA opening, venous pressure may increase to 50-60 mm Hg and higher, i. e. to the level of diastolic

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Received March 22, 2018; Accepted March 21, 2019; Published March 26, 2019

Citation: Vladimir E (2019) One_2_Eng_Sergei_Cancer. J Cancer Sci Ther 11: 094-095. doi: [10.4172/1948-5956.1000591](https://doi.org/10.4172/1948-5956.1000591)

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pressure plus the hydrostatic pressure of the liquid column. (I recall that in the veins the optimal pressure should be from 0 to 15-20 mm Hg, and in small arteries the diastolic pressure is not lower than 60-70 mm Hg.

Venous pressure in some veins is increased because the arterial blood, after the passage of AVA, is directed not only upward, but also down towards the small veins. This means that the pressure can spread up to the venules, especially in the most "comfortable" places: in the pelvic area and in the legs. This condition, when the veins pressure rises to values greater than 20-60 mm Hg., blocks completely or partially the capillary circulation and leads first to a decrease in the perfusion of tissues, and then to stasis, edema, fluid retention in the body, cancer and weight gain. Edema occurs because with increasing venous pressure, the permeability of the veins increases, so the amount of intercellular fluid also increases.

Thus, stopping blood circulation leads to some types of cancer. The same conclusion is given by the ancient Chinese theory of Qigong!

Of course, at the initial stage of the disease during the night rest in the horizontal position, the AVA closes normally. In this case, stagnant cold venous blood from the lower half of the body is again involved in a large circle of blood circulation. In young people, arterial and venous pressure for several hours of rest comes to its usual indicators. But in elderly people with excessive weight, most of the intercellular fluid remains in the body, despite abundant nycturia and sweating.

Simultaneously, with the development of pathology, i.e., with consequent damage of the valves of the veins, there is an irreversible circulatory disturbance, varicose of veins, then thrombosis. In other words, the development of circulatory insufficiency begins first in vulnerable areas: in the pelvic region and in the legs. Then, circulatory insufficiency may appear in the lungs, since in the lying position, the venous pressure along the body is almost the same. Venous blood of

a large circle of blood circulation can reach the lungs, the liquid can penetrate into the alveoli and block the small circle of blood circulation and the enrichment of the blood with oxygen. There are shortness of breath, high fatigue. The primary factor, as we see, was an increase of pressure in the veins and venules, and secondary - a malfunction of tissue perfusion, thrombosis.

But what does medicine say in this case? Medicine believes that venous plethora arises from the malfunction of outflow of venous blood due to the presence of some mechanical obstruction. These obstacles could be thrombi, embolus, tumor, etc. Therefore, with a critical increase in venous pressure, the blood circulation in the venules becomes irregular, but jerky or pendulum, with the reflection of the pulse wave from the blood clots.

Because of such reasoning, the consequence (the formation of stagnation and thrombosis) and the cause (increased venous pressure) in official medicine changed places. This was another mistake of cardiology. According to the new theory: first the venous pressure increases due to AVA, then come slowing of blood circulation, malfunction of perfusion of tissues, formation of blood clots. Simultaneously, to compensate this pathology, unsuccessful systemic neurohumoral adjustments begin. Thus, with the help of a new theory, it was possible to unite and logically justify all clinical observations during the development of so-called heart failure. Finally, I would like to conclude the following:

1. It is necessary to conduct a number of experiments to confirm a new theory of cardiovascular diseases, including cancer.
2. If the confirmation will take place, then new approaches to the prevention and treatment of CVD should be developed. It is necessary to develop equipment for searching for active ABA.
3. The author of the new theory is ready for cooperation.