Information on the Single Mechanism of Many CVDs Requires Top-Level Review

Ermoshkin Vladimir Ivanovich*

Physicist, Russian New University (RosNOU), Moscow, Russia

*Corresponding Author: Ermoshkin Vladimir Ivanovich, Physicist, Russian New University (RosNOU), Moscow, Russia. Received: June 11, 2020; Published: July 29, 2020

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. But there are no decisions yet on accepting this theory for consideration or on reasoned criticism of the New Theory. Let's look at this issue again.

Method: Exploring the many sources of information posted on the Internet. Discussion of proposed ideas at conferences, publication of original articles in Russian and English-language medical journals.

Results: First, we give a few brief established medical definitions of individual diseases of the cardiovascular system (CVS).

- By vegetovascular dystonia (VVD) or by "somatoform autonomic dysfunction of the nervous system" we understand pathology arising from a malfunction of the regulatory activity of the autonomic nervous system and manifested by various syndromes of dysfunction of the internal organs and systems (cardiovascular, respiratory, digestive and others). The prevalence of VVD is 25 - 40%, mainly among adolescents and young adults.
- 2. A paroxysmal tachycardia is an attack of an increase in the correct rhythm (usually 130 200 per minute) with a sudden start and end under the influence of pulses originating outside the sinus node. There are atrial forms (80% of all paroxysmal tachycardia), antrioventricular and ventricular. Paroxysmal tachycardia often occurs with various diseases of the heart and blood vessels, but at a young age it can also have a functional character.
- 3. Heart failure (HF) is an acute or chronic condition caused by a weakening of the myocardial contractility and congestion in the small (SCBC) or large circle of blood circulation (LCBC). It manifests itself as shortness of breath at rest or with a slight load, fatigue, edema, cyanosis. HF leads to the development of organ hypoxia. HF is one of the most common causes of human death. Increasingly, heart failure occurs in young and middle-aged people.
- 4. Violations of the outflow of venous blood from the brain and morning headaches. From the point of view of medicine, there are many reasons. These ailments can be both young and old.

Keywords: Cardiovascular System (CVS); Vegetovascular Dystonia (VVD); Heart Failure (HF)

As of June 2020, it is still believed that the basic mechanisms of all four groups of diseases have not yet been determined.

It seems that a modern person, starting from adolescence, is already sick and official medicine cannot always help. The new CVD Theory sheds light on a possible cause of these diseases, i.e. on attacks of paroxysmal tachycardia, on the nature of heart failure, on VVD on some others, because doctors, by their own admission, do not know a single reason, they talk about lists of dozens of practically the

Citation: Ermoshkin Vladimir Ivanovich. "Information on the Single Mechanism of Many CVDs Requires Top-Level Review". *EC Cardiology* 7.8 (2020): 49-53.

same "reasons". But this is not so, these are lists of factors, and the dominant mechanism of the onset of diseases, apparently, is one, and it remains unknown to doctors so far, although the mechanism is trivial. If you are guided by this mechanism, then you can begin to develop new methods of treatment and prevention of these diseases. The pairwise correlations of these and many other diseases indicate that there is one single cause, or rather, one mechanism that needs to be detected.

The author made an attempt to eliminate many misconceptions in modern medicine, in establishing the mechanism of the main diseases of the 20 - 21 centuries. You are to judge whether the author is right.

Let us briefly repeat the key points of the New Theory of CVD.

You can learn more about the New Theory in articles [1-15].

So, health problems in a person begin with periodic increases in blood pressure, which sometimes leads to forced discoveries in the vascular system of large shunts, or arteriovenous anastomoses (AVA). AVA usually opens for a short time, it is at these moments that a too high blood pressure drops, then, after a few seconds, the AVA closes again, and the blood pressure may rise again. At the time of AVA opening, blood from a high-pressure pool quickly flows into a low-pressure pool, i.e. into the veins. For example, from the superior mesenteric artery into the portal vein [1]. The figure for articles [2,3] shows ECG plots with jumps in blood pressure, which were obtained on the Russian "Cardiocode" device during the opening and closing of large AVAs.

What is the reason and meaning of AVA discoveries in a healthy person? The main reason is the presence of physical or psychological stresses, experiences, especially with a long time when a person is in a motionless, for example, a sitting or standing pose. The very opening of AVA gaps saves the cardiovascular system from overload, from excessive stretching of the walls of arteries, from premature strokes and heart attacks. In fact, AVA are valves that limit the increase in blood pressure above a certain value! But over time, many other problems arise, for example, excessive "deposition of blood" in the veins and organs, in addition, tissue edema, visceral obesity, as well as filling the space between adjacent organs with fluid, such as ascites, are possible.

As a result of blood transfusions imperceptible to the patient and the attending physician through the AVA, venous increases and its arterial volume decreases in the large circle of blood circulation (LCBC). From this time, the periodic organismic adjustments of the vascular lumens and blood volumes are forced to begin. Let me remind you that the volume of arterial blood in a healthy person is not half, but only about 15 - 20% of the total circulating blood volume. Blood pumping in both ventricles obeys the Frank-Starling law: an increase in the degree of stretching of the heart muscle leads to a greater contraction force. The strength of each heartbeat depends on the size of the venous inflow and is determined by the final diastolic length of the myocardial fibers. It is believed that the throughputs of the left and right halves of the heart are usually the same.

With frequent openings/closures of AVA, body compensation of volumes and regulation of venous blood pressure are a big problem for the cardiovascular system. Apparently, this is the reason for attacks of the type of vegetovascular dystonia (VVD). In both VVD and panic attacks, some brain cells can sense ischemia. At a young age, VSDs occur, then serious CVDs are possible as they grow older. AVA discoveries are tantamount to normal arterial bleeding, albeit insignificant. It is impossible to directly return excess blood from veins to arteries directly. The body can replenish the arterial pool of LCBC (large circle of blood circulation) either by increasing blood flow through the right ventricle, i.e. through the pulmonary circulation (SCBC), which is difficult to do, or by the reverse flow of intercellular fluid through the arterioles of the LCBC, which is more likely, but the question remains open. In practice, a partial solution to this issue has been known for a long time: periodic venous bloodletting or donation, leeches and Arabian hijama, but for some reason these methods are hushed up by medicine, to put it mildly. An invasive rescue option is theoretically possible, but it is very complex and expensive: regular dosed direct injections of your own venous blood into the arteries.

Citation: Ermoshkin Vladimir Ivanovich. "Information on the Single Mechanism of Many CVDs Requires Top-Level Review". *EC Cardiology* 7.8 (2020): 49-53.

We can assume that with a significant leakage of arterial blood into the venous pool and to maintain the necessary blood pressure, the arterial pool of the LCBC itself must be reduced. And since the arterial pool, unlike the venous, has no "high-pressure arterial depots", we get the result: a gradual spasm of the vessels of the peripheral organs in the form of cooling feet and hands. Further, other arteries enter the state of spasm. In general, a spasm of numerous arteries and arterioles due to loss of arterial blood - this is the forced reduction of the arterial pool at the cost of turning off the power of some organs. Lastly, spasm spreads to the vessels of the brain, since it has the highest priority.

So, with a decrease in arterial volume (multiple spasm of arterioles), in order not to damage the small and medium arteries, including the brain, with pressure, the shock volume of blood of the left ventricle is forced to decrease, because full stroke volume is possible only with open arterioles of almost all organs. We can assume that all of this is provided for by the organismic neurohumoral vegetative adjustments. But, on the other hand, since the venous pressure and the volume of venous blood of the LCBC exceeds the optimum, according to the Frank-Starling law, somewhat increased shock volumes of blood begin to be pumped through the right ventricle. Apparently, this imbalance between the emissions of the right and left ventricles leads to new problems: to blood retention in the lungs, shortness of breath, asthma, and even to possible pulmonary edema. Doctors usually diagnose heart failure of the left or right ventricles of the heart due to a decrease in myocardial pumping function. But reducing the stroke volume of the left ventricle is a physical necessity, not a disease!

Apparently, at the beginning of the development of this defect, the diagnosis of heart failure can be made with a completely healthy heart. Perhaps for the same reason, cardiomyopathy and thickening of the walls of the myocardium develop, because loss of arterial blood volume requires a decrease in the volume of the left ventricle.

Now about arrhythmia. For example, when an AVA is opened between a large artery and a large vein, blood volume increases in the vena cava, and excessive venous pressure spreads throughout the venous system: first down and then up. At the first stages of the situation with a blood transfusion into the veins, while the person is still young and healthy, while the venous valves well restrain excess volumes of additional blood and do not allow its distribution due to gravity down into the pelvic and leg areas, there is nothing pathological in the vessels not happening. The situation is saved by moderate physical activity and a daily 8-hour horizontal position of the body during sleep: at night in a horizontal position, the person's venous pressure is equalized throughout the body, arterial and venous blood volumes are gradually optimized. But together with the years lived, venous valves can be damaged due to an increase in venous blood volume. With a passive lifestyle, excess venous and tissue pressures rise higher and higher, most often the legs first suffer, then the pelvic organs and kidneys. With an excessive increase in venous pressure at the point where the hepatic vein flows into the vena cava, partial blocking of the liver, pancreas, etc. begins. Health begins to deteriorate rapidly, the volume of venous blood and intercellular fluid with a passive lifestyle can continue to increase further.

The main and most dangerous thing that can happen with an increase in venous blood volume is overflow of the vena cava and blockage of the liver. Due to a heart pulse spreading through the aorta and the hepatic artery, a liver overflowing with blood can begin to pulsate [15]. The pulsation is transmitted to the vena cava in contact with the liver, the reliability of this is confirmed by the simultaneous pulsation of the cervical veins. Through the vena cava, pulsation is transmitted to the right atrium. A mechanical wave of sufficient power can transfer to the myocardium. Further options are possible depending on previous damage to the heart, the presence of scars and tissue conductivity.

The first option: a mechanical wave from a vena cava directly in the atrium produces mechanically induced excitation of the sinus node, and an atrial tachycardia attack with narrow QRS teeth begins.

The second option: mechanically induced excitation is focused near the sinus node, and thereby excites cardiomyocytes (CMC), and then the sinus node. An atrial tachycardia attack with wider QRS teeth begins.

Citation: Ermoshkin Vladimir Ivanovich. "Information on the Single Mechanism of Many CVDs Requires Top-Level Review". *EC Cardiology* 7.8 (2020): 49-53.

The third option: a mechanical wave runs through the heart from top to bottom and focuses in the lower part of the myocardium. Ventricular CMCs are excited, an attack of ventricular tachycardia with very wide QRS teeth begins.

In all three cases, as the doctors say, "correct rhythms" with different heart rates are formed, but in fact they are all wrong, because cause myocardial damage due to ischemia! But the "correctness" of the rhythm and the sudden beginning and end of attacks are akin to the physical phenomenon of "resonance". The resonance also begins and ends suddenly, sometimes with great damage, even for solid, for example, metal structures.

According to the ECG, the distance between the R teeth on the ECG for all three options is too small, constant and independent of the phases of respiration, because conductive tissues are already too stressed. The work of the sinus node due to the excess refractory time of specialized cells compared to the pauses between strokes cannot begin. These cells are waiting for any pause to turn on, but it is not there. In fact, the accelerated heart rhythm is determined by the time the mechanical wave travels through the aorta, artery, liver, along a tense vena cava, along the myocardium up to a specific place of excitation. That is how an attack of paroxysmal tachycardia begins with an unusually high heart rate (HR): 130 - 200 beats per minute. In fact, the heart creates a pulse wave and with the same wave, running in a circle, excites itself many times in a row. Such attacks start and stop suddenly. Attacks can last from 1 - 3 hits in a row to several days. An attack can be relieved only by sufficient relaxation (or, conversely, an increasing spasm) of the vessels, the creation of barriers to mechanical impulses, or through special methods. It is interesting that official medicine in many such cases (including atrial or ventricular fibrillation) completely forgets about mechanically induced excitations and tells us about the "craziness" of the heart, about the inhibition of the automatism of the sinus node, about the increased automatism of certain cells, about the increased sensitivity of cells, about additional pathways, about new ectopic foci and about their drift. Well, who is right here: official medicine or the New Theory of CVD?

And in conclusion, a small paragraph on the causes of impaired venous outflow from the brain and morning headache.

In the lying position during night sleep from the lower half of the body, the outflow of excess venous blood and intercellular fluid accumulated while sitting and standing during the "working" day occurs. This is facilitated in the supine position by alignment of the middle venous pressure along the whole body. Gradually, in the morning, fluids flow into the upper half of the body, to the head. In fact, the outflow of venous blood from the brain in the morning is hindered by the "oncoming flow of dirty blood and tissue fluid" from the lower half of the body. Therefore, at 5 - 8 in the morning, many may experience morning headaches, unexplained increases in blood sugar at night and in the morning, and more serious events are also possible. This is confirmed by medical statistics.

Conclusion

- 1. The article provides a brief overview of the causes of the most dangerous human diseases disclosed in the New Theory of CVD.
- 2. In the review, the example of the occurrence of diseases such as heart failure, paroxysms of tachycardia, VVD, impaired venous blood outflow from the brain shows the general mechanism of many dangerous diseases. This mechanism is the stress and pathological work of AVA anastomoses.
- 3. It seems surprising to the author that for 9 years of publication of the New Theory of CVD in various Russian and English-language medical journals, the reaction of the leading structures of Russian medicine is zero. In official medicine, it continues to be believed that there are many causes of CVD and the underlying mechanism of their occurrence remains unknown.
- 4. Perhaps the leaders of Russian medicine, like all world medicine, have completely different tasks in priority: not preserving the lives of young and middle-aged people from sudden illnesses, not prolonging a person's life, but simply participating in the treatment and "correct" distribution of certain dividends? After all, the sale of a large number of tablets brings huge profits.

5. I'll propose carefully: a prolonged suppression of the results of the New Theory, apparently, confirms the existence of the theory of the "golden billion".

Bibliography

- 1. Internet resource. Clinical case. Russian.
- Lukyanchenko VA. "Cardiometric signs of performance of arteriovenous anastomosis in human cardiovascular system". *Cardiometry* 8 (2016): 22-25.
- 3. Ermoshkin VI. "New theory of arrhythmia. Conceptual substantiation of arrhythmia mechanisms". Cardiometry 8 (2016): 6-17.
- 4. Ermoshkin VI. "The new theory of cancer complements ancient Chinese Qigong therapy" (2017).
- 5. Ermoshkin V. "The Cause of Some Cancers because of the Open Arteriovenous Anastomoses". *Journal of Gastrointestinal Cancer Stromal Tumors* 2 (2017): 1000111.
- 6. Ermoshkin Vladimir Ivanovich. "New Theory of CVD/Cancer and Diabetes". EC Cardiology 7.1 (2020): 01-06.
- 7. Ermoshkin VI. "The new theory of heart failure". London (2017).
- 8. Vladimir Ermoshkin. "Multiple Sclerosis. Why Did The "Progressive Theory" of Paulo Zamboni Not Find Support? But Then A New one Was Born!". International Journal of Clinical and Medical Cases 2.1 (2018): 04-06.
- Ermoshkin VI. "Arteriovenous anastomoses and cardiovascular diseases". 8th Cardiovascular Nursing and Nurse Practitioners Meeting. Las Vegas, USA (2016).
- 10. Ermoshkin VI. "Venous congestion due to large arteriovenous anastomoses". 566 Chiswick High Road, London, Greater London, W4 5YA, United Kingdom (2016).
- 11. Ermoshkin VI. "Heart transplantation mysteriously eliminates arrhythmia". Cardiometry 8 (2016): 18-21.
- 12. Ermoshkin V. "Pathological Role of the "Invisible" Anastomoses". Journal of Bioengineering and Biomedical Sciences 6 (2016): 209.
- 13. Vladimir Ermoshkin. "Numerous studies show: not an excess of cholesterol, but first of all a systemic inflammation is the real reason of many cardiovascular diseases and of sudden death". *International Journal of Cardiology and Heart Health* 1.2 (2017): 22-24.
- 14. Vladimir Ermoshkin. "The pathological mechanism of systemic inflammation in humans has opened. The reason is uncontrolled leakage of arterial blood through arteriovenous anastomoses". *International Journal of Cardiology and Heart Health* 1.2 (2017): 12-14.
- 15. Internet resource. Russian.

Volume 7 Issue 8 August 2020 ©All rights reserved by Ermoshkin Vladimir Ivanovich.