

A New Theory of Certain Cardiovascular Diseases

Vladimir I. Ermoshkin*

Russian New University (RosNOU), Moscow, street Radio, 22, Russia

***Corresponding Author:** Vladimir I. Ermoshkin, Russian New University (RosNOU), Moscow, street Radio, 22, Russia.

Received: November 11, 2016; **Published:** November 18, 2016

Under the conditions of unknown causes most of cardiovascular disease and high sudden death rate of young people proposed a new theory.

Objective

An attempt to study the mechanism of arrhythmia and other cardiovascular disease (CVD) in humans.

Methods

I graduated from the Physics Department of Moscow State University in 1978. Since 2010 I have been studying cardiology in RosNOU. I participated several times at medical conferences in the People's Friendship University, St. Petersburg (Cardiostim-2016), Brisbane (Arrhythmia-2016), I had several discussions with leading Russian cardiologists.

Results

I found an error in the theoretical cardiology. This error is 100 years old. It turns out that when revealing the causes and mechanisms of cardiac arrhythmia the possibility of mechanical nature of cardiomyocytes excitation as an addition to the bioelectric nature excitations of the sinus node was not taken into account. In fairness, it should be noted that, for example, the group of Professor A.G. Kamkin came very close to solving this problem, but the decisive step was not done [1]. In the official medicine, it is considered that cardiac rhythm disorders occur mainly due to the micro and macro "reentry", "ectopic foci". It led to the situation when the official medicine cannot explain the cases of death caused by acute heart failure (AHF) or sudden cardiac death (SCD) in a young healthy heart in case of absence of acquired heart disease (according to autopsy reports). It turned out that a large arteriovenous anastomoses (AVA) may be periodically opened under the influence of stress or significant physical loads in humans [2] and the blood under high 120 - 80 mm Hg pressure, begins to flow into the veins, not reaching the destination, i.e., cells. It leads to arterial pressure jumps [3], at the same time the pressure drops in the arteries and increases in the veins. The overall volume of arterial blood reduces and the venous blood volume increases. Tests on the device "CARDIO-CODE" confirmed these phenomena [4].

Vena cava dilates and its tone increases, and pulse waves start going through the AVA along the vena cava to the neck veins, atria and ventricles of the heart [5]. Mechanical waves can run along the "sphere" of the heart and focus on both the atria and ventricular tissues. This is the solution of the problem of notorious ectopic foci. At a sufficiently high energy of mechanical tension in myocardial tissue may appear extrasystoles, or, under certain additional conditions, paroxysmal ventricular tachycardia from any point in the apex of the heart [6]. Atrial fibrillation is usually a result of ischemia and simultaneous excitations initiated in several areas of heart muscle, i.e., superposition of mechanical and electrical chaotic launches. So, ectopic foci are points of focusing the energy of mechanical waves, thus, the foci can easily move (drift), disappear and re-appear, depending on the concentration of mechanical tensions [7,8].

In young individuals with healthy heart such events occur almost without a trace. But frequent attacks of paroxysmal tachycardia and fibrillation can lead to progressive atherosclerosis and fibrosis of heart due to myocardial ischemia in case of increased heart rate. But that is not all. Arterial blood passed into the vena cava through opened AVA, where operating pressure is from 0 to 10 mm Hg, mixes with venous blood and then retrogradely flows along all small veins to venules leaking gradually into the interstitial fluid. In venules, the

pressure can be increased to approximately 20 - 30 mmHg. Due to the lack of value of the pressure gradient between the arterioles and venules, capillary blood flow stops or slows down, in certain organs and parts of the body cells are starving. In essence, it is a hydraulic lock in which the fluid cannot leave the small veins according to the laws of physics, and not because of “weak vessels” or “lack of potassium- magnesium” as doctors say. As a result, extremities temperature falls, edemata, varicose veins, stagnation of fluids appear. Lymphatic vessels also stop functioning in the affected organs. Typically, at a later stage due to edema of lung tissue and increased venous pressure in the systemic circulation a heart failure occurs. (Let us note that the lungs have the capillaries of both large and small blood circulations).

Generally speaking, due to gravity the edemata of tissues and organs are distributed in such a way that they first arise in the lower extremities and pelvis area, and then gradually rise up. Usually, at latter stages, pulmonary edemata may occur, as lungs are at the same level with the heart in terms of contribution of the fluids hydrostatic pressure. Excess edematous fluid leads to shortness of breath, spasm of the alveoli, blocking of oxygen enrichment of venous blood in the pulmonary circulation. AHF may occur even without infarction of the heart. Blockings of blood flow due to the opening AVAs can lead to diseases comorbidity in a human, leading a modern lifestyle without physical exercises. Daily exercises, regular sleep, massage, swimming, daily breathing exercises can serve a good prevention of circulatory disorders. Yes, just the breathing exercises, for example, based on the methods by Strelnikova, contribute to the diaphragm training. Just the trained diaphragm is the main working body for pumping the venous blood from the most remote places.

Recently, appeared one more indirect proof of the plausibility of the theory proposed by me. To know more about testing of the “revolutionary” method of struggle against hypertension read the reports [10,11]. The device ROX AV Coupler was used in the testing, it is developed by the American company ROX Medical. Tests were carried out in England in 2014 - 2015. This device is essentially an artificial implant (arteriovenous shunt) with a 4-mm hole diameter and a continuous throughput of about 800 ml/min. The shunt was placed between the iliac artery and the adjacent vein. Tests were conducted on hypertensive patients resistant to treatment drugs. Due to this method of treatment of resistant forms of hypertension it was a success to show that a reduction in systemic blood pressure by an average of 27/20 mmHg really occurred. Unfortunately, in 2 - 9 months over the distal shunt in 29% of cases appears a stagnation of venous blood and edemata, which are the same as in a natural permanently opened anastomosis [3,5]. One may ask why in the open natural arteriovenous anastomoses arrhythmias occur [2,3], and in permanently opened ROX-shunt do not, but on the contrary, as stated by researchers, the implant method contributes to treatment of not only arrhythmias, but some other CVDs [12].

In my opinion, it happens because the implant is made of solid material, which is significantly different from natural tissue of anastomoses, so the mechanical waves traveling along the walls of arteries are effectively reflected from foreign material and do not hit the vena cava wall. Thus, ROX Coupler reliably eliminates the cause of arrhythmias due to the impact of pulse waves. The ROX Coupler implant, struggling against the effects of hypertension, reduces the blood pressure and thereby eliminate the reason for the opening and closing of natural AVA. We can assume that after the installation of ROX-shunt the large natural AVA are forced to be in the closed state permanently, and the heart, lungs and some large vessels have to work with the increased load and it is useless to pump some extra volume of blood from major arteries into the veins. This excess value of pumping is 800 ml/min or 16% of the norm in 5000 ml/min. In my opinion, this solution is not optimal. It is obvious that for hypertensive patient in the future it is needed to develop the implants with a variable diameter of shunt lumen, down to zero value of lumen, depending on the current blood pressure in the arteries. But it is still better to “use” the natural anastomoses given to us by nature, and for that it is necessary to lead a healthy lifestyle with daily moderate physical loads on muscles and cardiovascular system. Another confirmation of the fact that the mechanical impulses is suppressed heterogeneity of tissue or a surgical suture is statistics on the results of greater efficiency bicaval (compared to biatrial) technique in heart transplant [13].

Conclusions

Unhealthy lifestyle, hypodynamia, opening and not closing in time AVAs can sometimes lead to a variety of CVD. To avoid the cardiac arrhythmias attacks and prevent the SCD it is necessary to somehow suppress the mechanical waves running along one and the same circle: aorta-mesenteric artery- AVA - portal vein - vena cava – atrium - ventricle. That is why there are extraordinary heartbeat intervals

with the same clutch. That is why the extraordinary heartbeats occur with equal intervals between linkages. And that is why in case of tachycardia the heartbeats occur with equal intervals between adjacent beats and there is no heart rate modulation caused by the respiratory phase change. It seems a re-entry phenomenon has mostly a mechanical nature. The new theory can logically explain the etiology of cardiovascular diseases, that is still impossible to do by means of official theories. Perhaps, the method of radiofrequency ablation (RFA) of the myocardium, that is so popular today, will be recognized as not the best solution, and at least partially replaced by the suppressors of mechanical waves in concrete vessels (arteries or veins [9]).

I believe that it is necessary to conduct a series of field experiments in order to prove the new theory.

Bibliography

1. Kamkin AG., *et al.* "Fibrillations, defibrillations". *Nature* 4 (2002).
2. <http://valsalva.ru/viewtopic.php?t=1101&sid=137874936ec435e6be6626bf749f6a0f>
3. Lukyanchenko VA. "Cardiometric signs of performance of arteriovenous anastomosis in human cardiovascular system". *Cardiometry* 8 (2016): 22-25.
4. Vladimir A Lukyachenko. "Mechanisms of high heart rate". *Cardiometry* 8 (2016): 26-30.
5. Vladimir I. Ermoshkin. "New theory of arrhythmia. Conceptual substantiation of arrhythmia mechanisms". *Cardiometry* 8 (2016): 6-17.
6. Arrhythmias Conference Materials. Australia, Brisbane (2016).
7. Ermoshkin VI. "New hypothesis of cardiac arrhythmias in humans". *Cardiostim* (2016).
8. Ermoshkin VI. "Arteriovenous anastomoses and cardiovascular diseases". 8th Cardiovascular Nursing & Nurse Practitioners Meeting, Las Vegas, USA (2016).
9. Ermoshkin VI. "Suppressor pathological venous pulse wave to prevent arrhythmias and sudden cardiac death. Stage: Idea; Biotech and medicine". Moscow (2016).
10. Melvin D Lobo., *et al.* "Central arteriovenous anastomosis for the treatment of patients with uncontrolled hypertension (the ROX CONTROL HTN study): a randomised controlled trial". *The Lancet* 385.9938 (2015): 1634-1641.
11. Kapil Vikas., *et al.* "Central Iliac Arteriovenous Anastomosis for Hypertension: Targeting Mechanical Aspects Of The Circulation". *Current Hypertension Reports* 17.9 (2015): 585.
12. Rodney Brenneman., *et al.* "Methods, systems and devices for treating cardiac arrhythmias". *Freshpatents* (2016).
13. Vladimir I. Ermoshkin. "Heart transplantation mysteriously eliminates arrhythmia". *Cardiometry* 8 (2016): 18-21.

Volume 2 Issue 5 November 2016

© All rights reserved by Vladimir I. Ermoshkin.