

Arteriovenous Anastomoses and Cardiovascular Diseases

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Abstract

Aim: *Why are most of the cardiovascular diseases (CVD) with unknown etiology? An attempt to solve the riddle, An attempt to study the unknown role of the arteriovenous anastomoses (AVA), an attempt to finally clarify the causes of cardiac arrhythmia.*

Methods: *Medical consultations, information search in literature, participation in medical conferences, correspondence with scientists, discussions with Russian leading cardiologists.*

Results: *The official medicine: arteriovenous anastomosis (fistulas) - an abnormal connection between an artery and a vein. Normally, blood flows from arteries into the capillaries and then into the veins. Most cardiologists agree that the role of small or large AVA in human circulatory system is still poorly understood. We have found that along with the positive role, the anastomoses, especially large AVA, periodically have pathological effects on the cardiovascular system. The device "Cardiocode" was used for testing. Large arteriovenous anastomoses (AVA) can be opened under the influence of stress or physical loads. Periodically, the pressure change in the arteries and veins. Vena cava expands, its wall's tone increases and pulse waves start to path through the AVA along the elastic walls of the vena cava to the right atrium and to the neck veins. Mechanical impulses can excite heart from various points of the atria or ventricles, disrupting the sinus rhythm. The result is the following: extrasystoles appear, tachycardia attacks, at the same time the blood flow is blocked on almost all the peripheral segments of circulatory system, edemata appear. Increased venous pressure stops the capillary circulation, which eventually leads to heart failure, even in a healthy heart. Severe metabolic disorders, it appears that leads to disease comorbidity, to venous congestion, to disease pelvic, to heart failure, to sudden cardiac death (SCD).*

Conclusions: *I think we are approaching in the understanding of CVD. The absence of special breathing exercises, drinking large amounts of beer, smoking, lack of exercise and presence of large AVA can sometimes lead to a variety of diseases, to metabolic diseases. To get rid of the attacks of cardiac arrhythmias and the prevention of SCD we need to find some way to suppress the mechanical waves running through AVA, as "reentry" phenomenon has a mechanical nature. It is necessary to continue studying the AVA to develop new measures for neutralizing the pathological events associated with the open AVA.*

Keywords: Arteriovenous anastomosis, Arrhythmia, sudden cardiac death, Metabolic diseases, Reentry.

Introduction

Theorists and practitioners in Cardiology have written mountains of articles, but still with an incomplete use, because there is a fundamental shocking error in theoretical cardiology for 100 years! This article is a new point of view. In the official cardiology, especially during the last 30-50 years, there have been different trends in approaches to the solution of this important problem for everyone. In the majority of complex cases, often doctors recommend RFA and ICD, i.e. radiofrequency ablation and implantable cardioverter-defibrillators. Heart diseases are now

the leading cause of death. But there is another way of cardiology development and related medical disciplines. This promising way will be discussed in this article.

Methods

Consultation and discussions, finding information via Internet about possible causes of arrhythmias, comorbidity for certain diseases, studying classical and contemporary primary relevant sources, participation and presentation at medical conferences, discussions of theoretical and clinical issues with health care experts Russian Academicians Evgeny Chazov, Amiran Revishvili, Professors E. Shirokov, V. Shirinsky, Doctor M. Rudenko and others laid the groundwork for the materials and methods of this paper. In

fairness, it should be noted that not all of the above experts are currently fully support the basic idea of the theory proposed by the author herein. During the discussions, I was asked a number of fundamental questions to which, I think, I managed to find eligible logical answers.

Results

The modern concept of arrhythmia and sudden cardiac death based mainly on violations of homeostasis of internal organs and the cardiovascular system: deficiency of magnesium, potassium, omega-3, coenzyme Q10 and L-carnitine, activity and angiotensin converting enzyme of catecholamine, which can lead to electrical instability of myocardium. This concept partly is valid, but insufficient.

We can conclude that all modern medical theories believed that CVD causes lie at micro levels. We assume, there is some success in modern medicine, but it is necessary to do a lot in the near future. Generally speaking, in cardiology, great problems have existed for a long time, such as high mortality from cardiovascular diseases and the high probability of sudden cardiac death in able-bodied age population.

Specifically, unsolved problems include:

- Arrhythmia causality,
- Sudden cardiac death,
- Diseases comorbidity,
- Cardiac insufficiency causality in a healthy heart,
- Overweight and obesity,
- Venous thrombosis,
- Bronchial asthma,
- As well as many others.

In 2011 I became interested in the problem of arrhythmia. Being a physicist, and an engineer, I transferred my knowledge, my experience, my new ideas to human body, as the laws of physics are the same for all substances. That same year I published the hypothesis of arrhythmia based on opening and inadequate closing time of the large arteriovenous anastomoses (AVA).

In 2011-2016 I frequently highlighted my new theory an international medical conferences including 5 times at Peoples' Friendship University of Russia in Moscow. I met and corresponded with Russian academics in cardiology. Of course, there appeared questions, discussions and negations of my theory. But, as to my mind, I gave logically substantiated answers to all the questions. Most recently, I took part in two medical conferences arrhythmia problem: in St. Petersburg and in Brisbane [1,2].

I think that the New Theory of Arrhythmia (NTA) has been asserting itself for the last 5 years. In the process of study and improvement of my theory, new knowledge appeared on the additional mechanism of maintaining the necessary pressure in arteries and the arrhythmia of heart.

In 2015, with the use of the Cardiocode device, we confirmed that

acute pressure jumps in patients are caused by the opening and closing of the large AVAs [3]. These AVAs are intended to prevent an extreme rise in arterial pressure both on local and system levels. This can put pressure in CVS which can lead to dangerous stress on vessel walls, stroke and infarction of the organs.

In healthy, moderately fit individuals the AVAs functions correctly. But, in case of hypodynamia, overeating, and an unhealthy lifestyle, the process of the AVA's ability to open and close is damaged. The AVAs lose their elasticity, cannot close and remain open indefinitely because of stress, stretching and deformation of the diaphragm walls and muscles, a big load on the liver, and absence of physical training. Even a small gap in the AVAs leads to problems in circulation. However, with muscular and breathing exercises, the diaphragm performance improves and the AVA's functions are restored.

When AVA are open and blood flows from the arteries to the veins. Pressure in the arteries decreases from 130-70 and the venous system increases between 0 to 10 mm Hg and higher. If the AVA does not close the vena cava overfills, its walls stretch, venous blood volume increases and the arterial one decreases leading the arterial pulse to pass through the AVAs along the venous walls up to atria, ventricles and neck veins. There are some grounds to state that the most frequent trajectory of the pulse passing is the following: aorta- superior mesenteric artery-AVA-portal vein-vena cava-atria-ventricles. Owing to the mechanical effect on the cardiomyocytes they can react and launch the excitation of the whole heart [4].

On health forums in internet a lot of patients indicate a connection between pressure fluctuations in the vessels of stomach and the beginning of an attack of arrhythmia. Usually, after a heavy meal there are pulsations, in a few minutes (or seconds), there are attacks of cardiac arrhythmias (posts on this issue a lot):

<http://www.medhelp.org/posts/Heart-Rhythm/Vagus-Nerve-and-Arrhythmia/show/344956> [English]
<http://www.healingwell.com/community/default.aspx?f=45&m=308402> [English]
<http://www.ibsgroup.org/forums/topic/46647-stomach-flutters-and-very-strong-pulsing-in-tummy/> [English]
<http://www.3630363.ru/?p=134616> (Irina, "Pulsation in the stomach...") [Russian]
<http://www.infarkt.ru/guest?p=84> (Elena, 10.12.2014, from the words: "Hello.I'm 45..."), [Russian]
<http://forum.antivsd.ru/index.php?action=printpage;topic=11692.0> [Russian]

Note that while the official medicine does not pay any attention to the patients clues about precursors arrhythmia.

Atherosclerosis increases the hardness of the vessel walls, so the shock amplitude is also increased. If the mechanical wave (short impulse) from any source effects the myocardium at the period after the top of T-wave can appear the extra systole, tachycardia,

fibrillation and even SCD [3]. The QRS profile of such a mechanical excitation may significantly differ from QRS when launching from the cardiac conduction system, the waves dilate, their polarity changes [5,6]. Thus, the heart reacts with the same efficiency to both the electrical and mechanical impulses. Reentry phenomenon is mainly mechanical nature. That is why the short-time precordial punch for restoration of the heart sinus rhythm was so popular in everyday practice for reanimation of a patient.

In addition, scar formation on vascular or myocardium leads to suppression of mechanical waves and reduces arrhythmia. This happens with RFA and heart transplants [7]. As a result, ventricular arrhythmia mysteriously disappears after a heart transplant. But, the incorrectly functioning AVAs lead to not only cardiac arrhythmias. The opened AVAs lead to the blocking of blood circulation in various organs.

How does blood circulation blocking and edemas occur?

It occurs according to the hydrodynamics laws taking into account the humoral and nervous regulations. The blood flows along the vessels from higher pressure segments to lower pressure segments. The greater the difference, the faster the flow. When the arterial blood appears in vena cava, where the pressure is significantly lower than the arterial one, it flows all over the vein in different directions: down, up, and to all the peripheral large and small veins. The veins, being a capacitance vessel with inelastic walls, are subjected to significant stretches, they cannot regulate the blood flow, they can only accumulate the blood. As a result, the arterial blood, passing through AVAs and mixing up with the venous blood, can flow into the veins of any organ, reach venules and penetrate into the tissue space. The quantity of intercellular fluid and the fluid located in the cavities may increase up to significant values with age. The dilation of the space filled with fluids is only limited by stretched fascia, skin and muscles.

The blood that has passed through AVA with pressure about 50-70 mm Hg flows to the venous channel segments with significantly lower pressure, about 0-10 mm Hg. The venous valves in the legs may be damaged owing to a great pressure difference taking into account the hydrostatic pressure [8]. In the case of the opened AVAs the difference between the pressure in arterioles and venules becomes insufficient, the cellular nutrition in the organs becomes slower, it actually stops, and no massage or foot warm-up can effectively start the blood circulation in legs. Only a forced closure of the AVAs, which may be located in other parts of the body, can help.

How to rapidly close the AVAs?

It may be that nobody knows how to do it as it is necessary to find the AVAs opening. Incorrectly functioning AVAs are the cause of circulation stagnation, overfilling of intercellular space with fluid, edema of organs, firstly in the lower parts of the body, then higher and higher. Such edemas are called right-side, as they are considered to be caused by right-side insufficiency of heart.

Help may also be special breathing exercises to train the diaphragm.

For example, after full inhalation, by the waves of negative pressure due to sudden movements of the diaphragm up through row partial exhales contribute to the outflow of stagnant venous blood from the small veins and venules in a large vein (further in hollow veins). Then, after a short pause, by the waves of positive pressure due to sudden movements of the diaphragm down during incomplete chain of sharp breaths help reverse the inflow of venous blood. Apparently, enough to make a row with 8 short exhales and then row with 8 short breaths. Such fluctuations total volume of venous blood on a daily basis, say for 10-20 minutes, can lead to the elimination of edema. But such exercises can be recommended only for patients who are in the early stages of the disease, but in thrombosis veins it needs caution and treatment under medical supervision.

How does left-side insufficiency in heart complicated with edema in lungs?

Conventional medicine states that the insufficiency is caused by the primary lesion of myocardium, chronic overload of the ventricle, disturbance of Starlings balance [9,10]. It is considered that the tactics of the best treatment is finding the primary disease. But the primary disease is difficult to find.

I have another opinion. In addition to the above mentioned causes the main cause is again the opened AVAs. It is known that during heart attacks occur pulsation carotid vein. Place ripple fixation is usually above the heart by 100 mm (+8 mm Hg for the right atrium). Carotid vein pulsations appear under attacks. Just the pressure increase in vena cava near the right atrium leads to a forced adaptation of the right half of heart to pathologic changes in tricuspid and pulmonary valve.

On the other hand, it is necessary to consider the following. The lungs have a double capillary network, one belongs to the systemic circulation and nourishes the lungs with oxygen and energy, taking the products of metabolism, and the other belongs to lesser circulation and is used for oxygenation (displacement of carbon dioxide from venous blood and its saturation with oxygen). Apparently, this venous blood from the systemic circulation penetrates into intercellular fluid in lungs and elevates the pressure in this fluid. Then the fluid penetrates into the alveoli and disrupts the process of oxygenation in the lesser circulation. As a result, an acute heart failure with shortness of breath, pulmonary edema and a possible SCD occur.

In medicine it is considered that the right-side insufficiency occurs before the left-side. The NTA proves it: because of gravity the edemas occur firstly in the lower limbs, then go up along the body and finally, in the last stage, the pulmonary edema occurs as the lungs are situated higher than all other internal organs. Thus, the mechanism of the right-and left-side heart insufficiency is the same: an excess venous pressure in the entire system due to the opened AVAs [3]. Similar effects, albeit with some peculiarities, may occur in the circulatory system of brain and head resulting in increased venous pressure, the same edemas, the same slowing of cellular nutrition. But from the point of view of hydromechanics,

there are some differences, as a head is situated above a heart. This is a topic of a separate study.

Points Against The New Theory

«... The property has a conductivity of only muscle tissue, which is not represented in the walls of blood vessels, so the assumption that the propagation of the pulse wave of the blood vessels to the heart are wrong. The response from the baro receptors that increase the heart rate, comes from the vascular center in the brain stem, so it is mediated process, with no direct link «vessel-heart.»

Moscow Scientific Center of Cardiovascular Surgery named Bakulev AN, PhD Filatov AG.

«...Your statements - speculative and incorrect. At the heart of cardiac arrhythmias are a variety of complex electrophysiological processes that are realized in the structures of the heart, we described and proved by numerous experimental and clinical studies. Your hypothesis is refuted by the development of cardiac arrhythmias in isolated hearts (surgically separated from the vascular system), as well as the occurrence of paroxysmal tachycardia without a pulse. » Russian Cardiology Research and Production Complex, Professor Golitsyn SP.

«...Your manuscript lacks original data supporting your suppositions. The current understanding of cardiovascular physiology and pathophysiology is much more sophisticated than you presume...

» Anne A. Knowlton, M.D. Editor OMICS Publishing Group/Clinical.

Comments

«...I have read your article with interest - it really presented original ideas, and for the scientist to introduce new ideas are always useful, it allows us to take a fresh look at the seemingly immutable truth. But any hypothesis needs to be discussed, a critical consideration - because in order to do something to check, you must have a good reason. The essence voiced by your idea is that “beats and tachycardia attacks sometimes occur in humans, including a fully healthy, the propagation of the arterial pulse along the contour of the vessels’ arteries - natural shunts (anastomoses) - veins” and due to the penetration of the pulse wave on the left and \ or the right atrium. “ You’re right that beats may occur from mechanical impact on the atrium or ventricle of the heart healthy. However, the heart is arranged in the chest in such a way as to minimize the mechanical effects of other tissues, in addition, it is enclosed in its own shell (the pericardium). You are right that the risk of premature beats in the hypertrophied heart above.

...» Institute of Experimental Cardiology, Professors Kapelko VI, Shirinsky VP.

«The author of a hypothesis is not a medical specialist. He is a physicist. It is possible that this fact explains his point of view on the mechanism of arrhythmia. Obviously, pathophysiology requires fundamental knowledge. This is especially important for explaining dynamic processes, such as the heart working. The assumptions of the author about the part in the pathogenesis of arrhythmia pulse wave appear convincing and reasonable. These assumptions require confirmation in the experiment, but every hypothesis only becomes a theory. The assumptions of the author

about the nature of ventricular fibrillation of the heart can be useful for the development of new treatment methods. The hypothesis may become a basis for deeper theoretical studies. I believe that the hypothesis is worthy of publication as material for discussion...» Head of “STOP Stroke” service Moscow, Professor Shirokov EA. «... I hope that your ideas will be of practical value.» Russian Academician Amiran Revishvili.

Conclusion

The main cause of many cardiovascular diseases is opening and inadequate closing time of arteriovenous anastomoses (AVA), which lead to blocking the blood circulation and stagnation of blood. The new theory cannot exclude SCD in a completely healthy heart as just the overfilled veins caused by opened AVAs lead to both the pulse wave passes and edemas of organs. The energy of mechanical punches into myocardium, when the pulse waves are passing, increases, as the atherosclerosis and fibrosis develop, and it is proved in practice: probability of arrhythmia attacks increases with age. In my opinion, the New Theory of Arrhythmia (NTA) has found and continues finding the logical interpretation to not only separate clinical cardiovascular cases but to many diseases considered to be nosological units before.

The New Theory of Arrhythmia has preliminarily substantiated two types of sudden cardiac death (SCD):

- A) The case of polytopic arrhythmia, which transfers into the ventricular fibrillation (especially in case of acute ischemia, infarction, mechanical effect),
- B) The case of acute heart failure with short breath and rapid edema in lungs transferring into fibrillations due to ischemia.

The situation occurring in the cardiovascular system with opened anastomoses (AVA) is similar to pumping a ball with a small hole in it. The ball will never be pumped in this instance. The same is true with the heart, if open AVA. It can eject sufficient volume of blood to aorta and further to cava vena, it works hard but inefficiently, and harms the whole body. The volume of arterial blood decreases critical. Many clinical events in cardiovascular diseases were logically connected and follow each other. In my mind, it is necessary to organize a number of “round table” discussions, experiments to affirm and expand the new theory, development of new treatment methods. The author of the present report is glad to cooperate.

Abbreviations

RFA: Radiofrequency Ablation; CVD: Cardiovascular Disease; CMC: Cardiomyocytes; CCS: Cardiac Conduction System; SCD: Sudden Cardiac Death; AVA: Arteriovenous Anastomose; CVS: Cardiovascular System; ES: Extrasystole; NTA: New Theory of Arrhythmia; IHD: Ischemic Heart Disease.

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